

A 28-year-old woman comes to the office due to vaginal spotting. The patient normally has regular monthly menses with 3-4 days of moderate bleeding. However, she has had spotting for the past 2 days, and her last menstrual period was 6 weeks ago. Vital signs are normal. Serum quantitative β -hCG level is 800 IU/mL (normal at 6 weeks gestation: 1,080-56,500), and a repeat level performed 2 days later shows an abnormal rise to 900 IU/mL. Pelvic ultrasound reveals no gestational sac in the uterus. If uterine curettage is performed, which of the following microscopic findings would most likely be found in this patient?

- ☐

A. Atypical endometrial cells, disorganized glands, and multiple mitoses
- ☐

B. Dilated, coiled endometrial glands and edematous stroma
- ☐

C. Inflammatory infiltration of endometrial glands
- ☐

D. Straight, short endometrial glands and compact stroma

Submit

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- ☐

A. Atypical endometrial cells, disorganized glands, and multiple mitoses (35%)
- ☒

B. Dilated, coiled endometrial glands and edematous stroma (50%)
- ☐

C. Inflammatory infiltration of endometrial glands (5%)
- ☐

D. Straight, short endometrial glands and compact stroma (8%)

Correct

50%

Answered correctly

02 mins, 58 secs

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Explanation

Endometrium with pregnancy-related changes

Decidualized stroma*

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This patient with vaginal bleeding, positive β -hCG, and no visible intrauterine gestational sac has a **pregnancy of unknown location**, which is concerning for an **ectopic pregnancy** (ie, embryo implantation in an extrauterine location such as the fallopian tube). Early ectopic pregnancy may not be visible on ultrasound; therefore, patients require serial β -hCG measurements for diagnosis.

This patient has an **abnormally low β -hCG level** for gestational age (eg, 800 IU/mL at 6 weeks gestation) plus an **inappropriate rise in β -hCG level**. In normal pregnancies, these levels double approximately every 48 hours; in cases of spontaneous abortion, levels typically decrease. Therefore, the most likely cause of this patient's low β -hCG level is ectopic pregnancy. Risk factors include pelvic inflammatory disease and prior pelvic surgery.

Despite its extrauterine location, an ectopic pregnancy changes the **uterine endometrium** due to the secretion of β -hCG, which signals the ovarian corpus luteum to continue progesterone production. Progesterone promotes endometrial remodeling and **decidualization**, which normally optimizes the intrauterine environment for pregnancy. Therefore, if uterine curettage is performed on this patient, it would likely show dilated, **coiled endometrial glands** and vascularized, **edematous stroma** (ie, decidualization) but no products of conception (eg, villi).

(Choice A) Atypical endometrial cells with disorganized glands and multiple mitoses are consistent with **endometrial adenocarcinoma**, a uterine malignancy that usually presents with vaginal bleeding. However, it typically affects postmenopausal women and is not associated with positive β -hCG levels.

(Choice C) Inflammatory infiltration of endometrial glands suggests **endometritis**, an infection of the uterine decidua. Endometritis is not associated with abnormal β -hCG levels.

(Choice D) Straight, short endometrial glands with compact stroma are found in the early **proliferative phase** of the menstrual cycle (ie, 4-7 days following menses, prior to ovulation).

Educational objective:
Ectopic pregnancy should be suspected in patients with a pregnancy of unknown location (ie, no visible intrauterine gestation) and abnormally rising β -hCG levels. Despite their extrauterine location, ectopic pregnancies promote

require serial β -hCG measurements for diagnosis.

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Despite its extrauterine location, an ectopic pregnancy changes the **uterine endometrium** due to the secretion of β -hCG, which signals the ovarian corpus luteum to continue progesterone production. Progesterone promotes endometrial remodeling and **decidualization**, which normally optimizes the intrauterine environment for pregnancy. Therefore, if uterine curettage is performed on this patient, it would likely show dilated, **coiled endometrial glands** and vascularized, **edematous stroma** (ie, decidualization) but no products of conception (eg, villi).

(Choice A) Atypical endometrial cells with disorganized glands and multiple mitoses are consistent with [endometrial adenocarcinoma](#), a uterine malignancy that usually presents with vaginal bleeding. However, it typically affects postmenopausal women and is not associated with positive β -hCG levels.

(Choice C) Inflammatory infiltration of endometrial glands suggests [endometritis](#), an infection of the uterine decidua. Endometritis is not associated with abnormal β -hCG levels.

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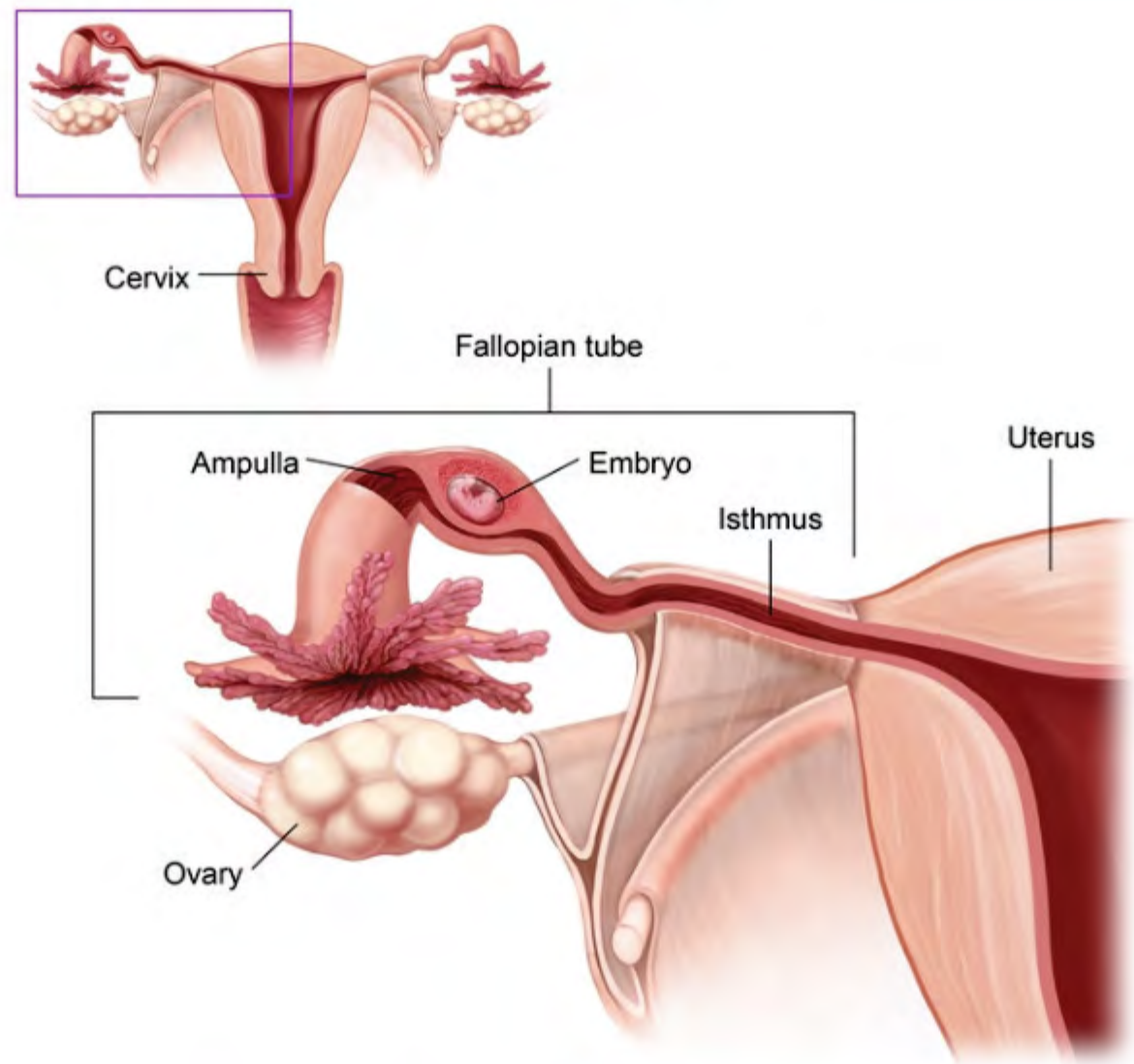
Educational objective:

Ectopic pregnancy should be suspected in patients with a pregnancy of unknown location (ie, no visible intrauterine gestation) and abnormally rising β -hCG levels. Despite their extrauterine location, ectopic pregnancies promote endometrial decidualization, which appears as dilated, coiled endometrial glands and vascularized, edematous stroma.

Reference

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Ectopic pregnancy



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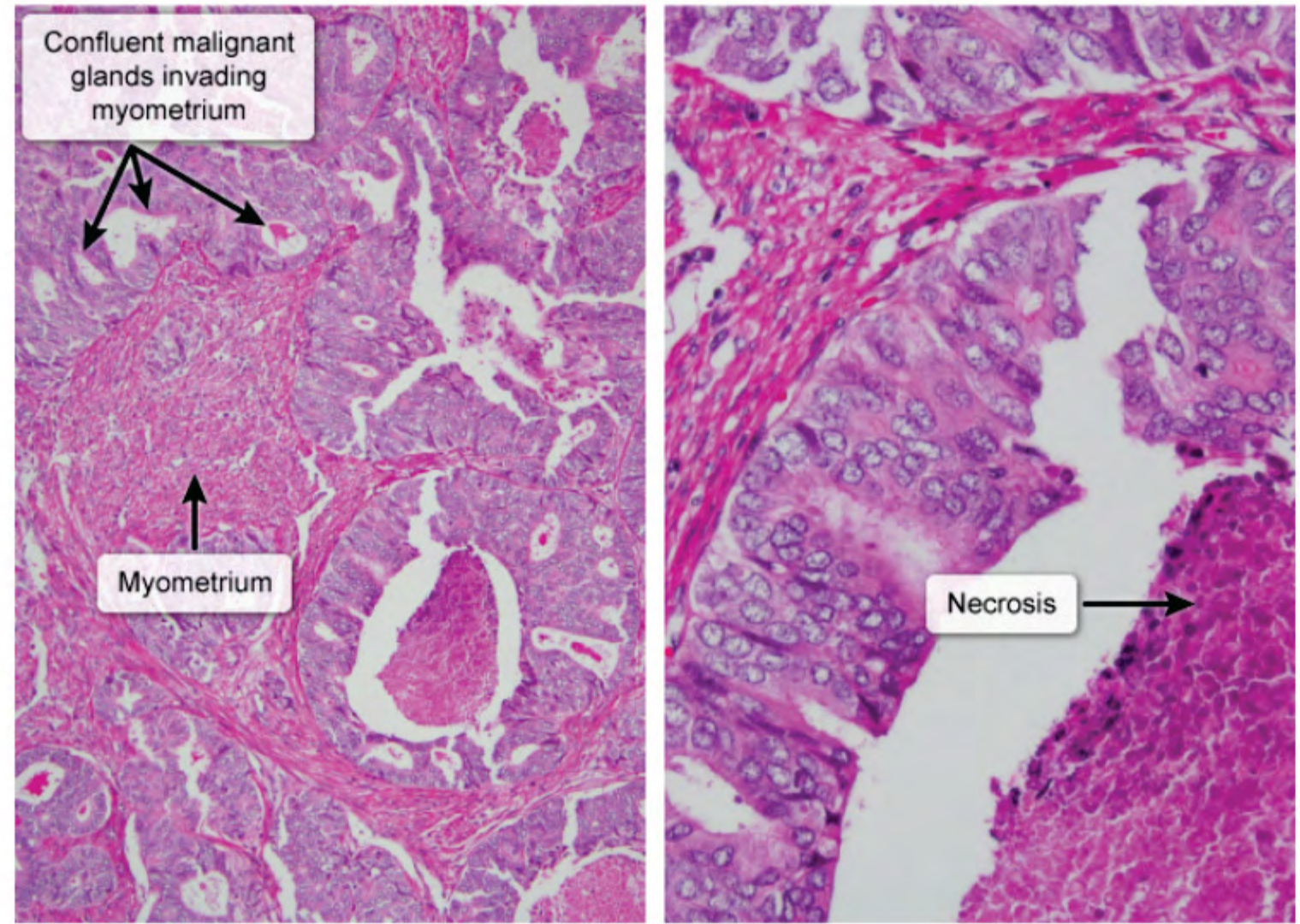
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Endometrioid endometrial adenocarcinoma



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Acute endometritis

Endometrial gland

Microabscess

Neutrophils

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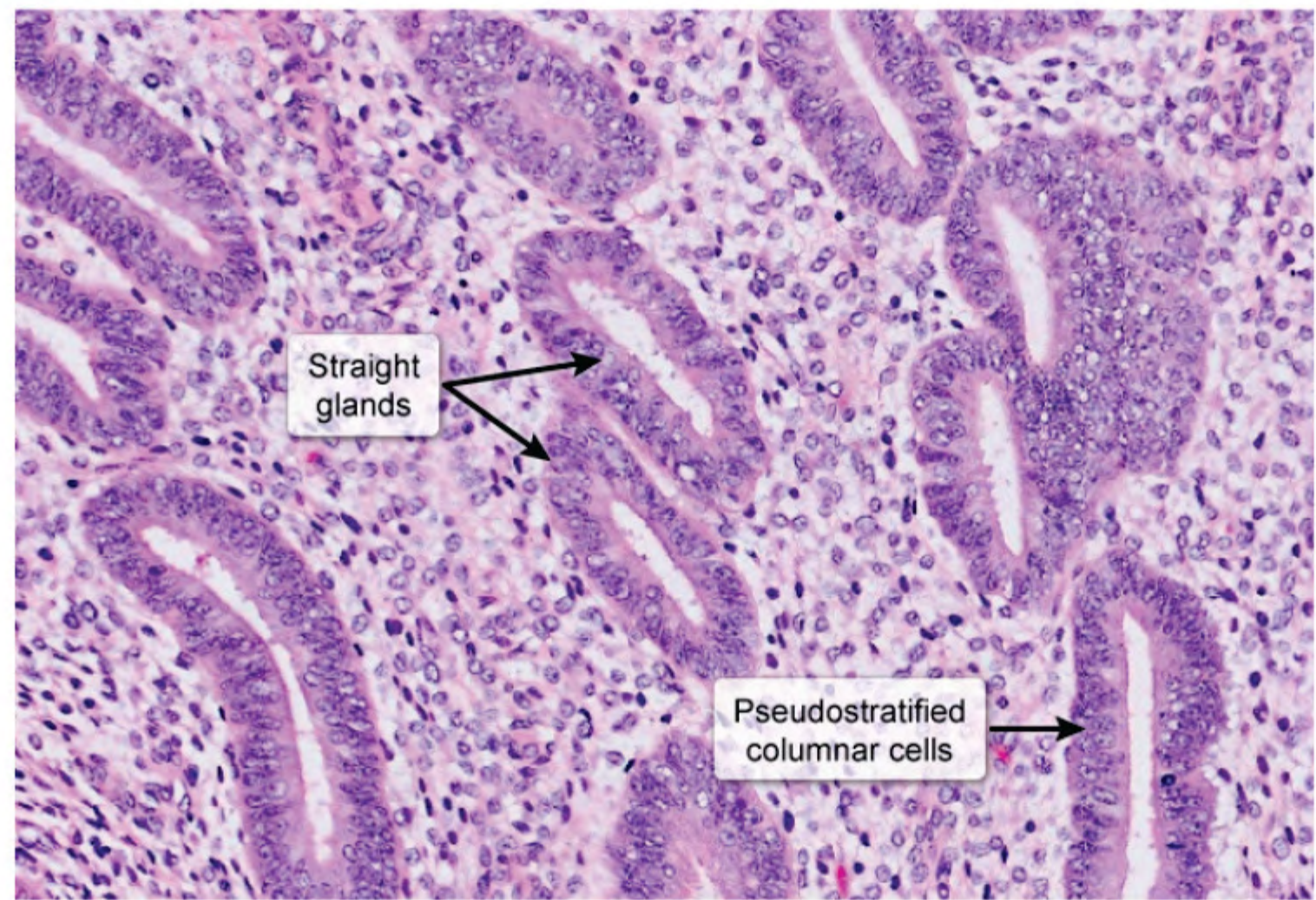
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Proliferative phase endometrium



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A 38-year-old woman, gravida 3 para 2, at 35 weeks gestation comes to the emergency department due to sudden-onset visual changes and headache. The patient reports "spots" in her vision for the past few hours and an occipital headache. She took a dose of acetaminophen, but it did not improve her symptoms. The patient's pregnancy has been complicated by gestational diabetes mellitus requiring insulin. Blood pressure is 168/114 mm Hg and pulse is 90/min. The pupils are equal and reactive to light. Extraocular movements are intact. Visual field testing shows partial loss of vision bilaterally. Which of the following is the most likely cause of this patient's symptoms?

- ☐ A. Choroidal neovascularization
- ☐ B. Decreased anterior chamber angle
- ☐ C. Hemorrhage into the vitreous humor
- ☐ D. Inflammatory optic nerve demyelination
- ☐ E. Retinal artery vasospasm

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- A. Choroidal neovascularization (18%)

✗

B. Decreased anterior chamber angle (8%)

C. Hemorrhage into the vitreous humor (26%)

D. Inflammatory optic nerve demyelination (5%)

✓

E. Retinal artery vasospasm (41%)

Incorrect

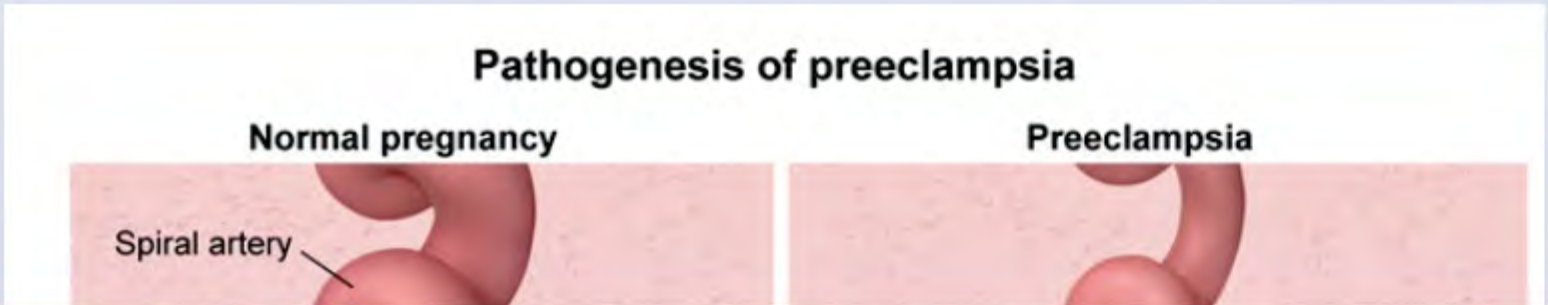
Correct answer
E

41%
Answered correctly

05 secs
Time Spent

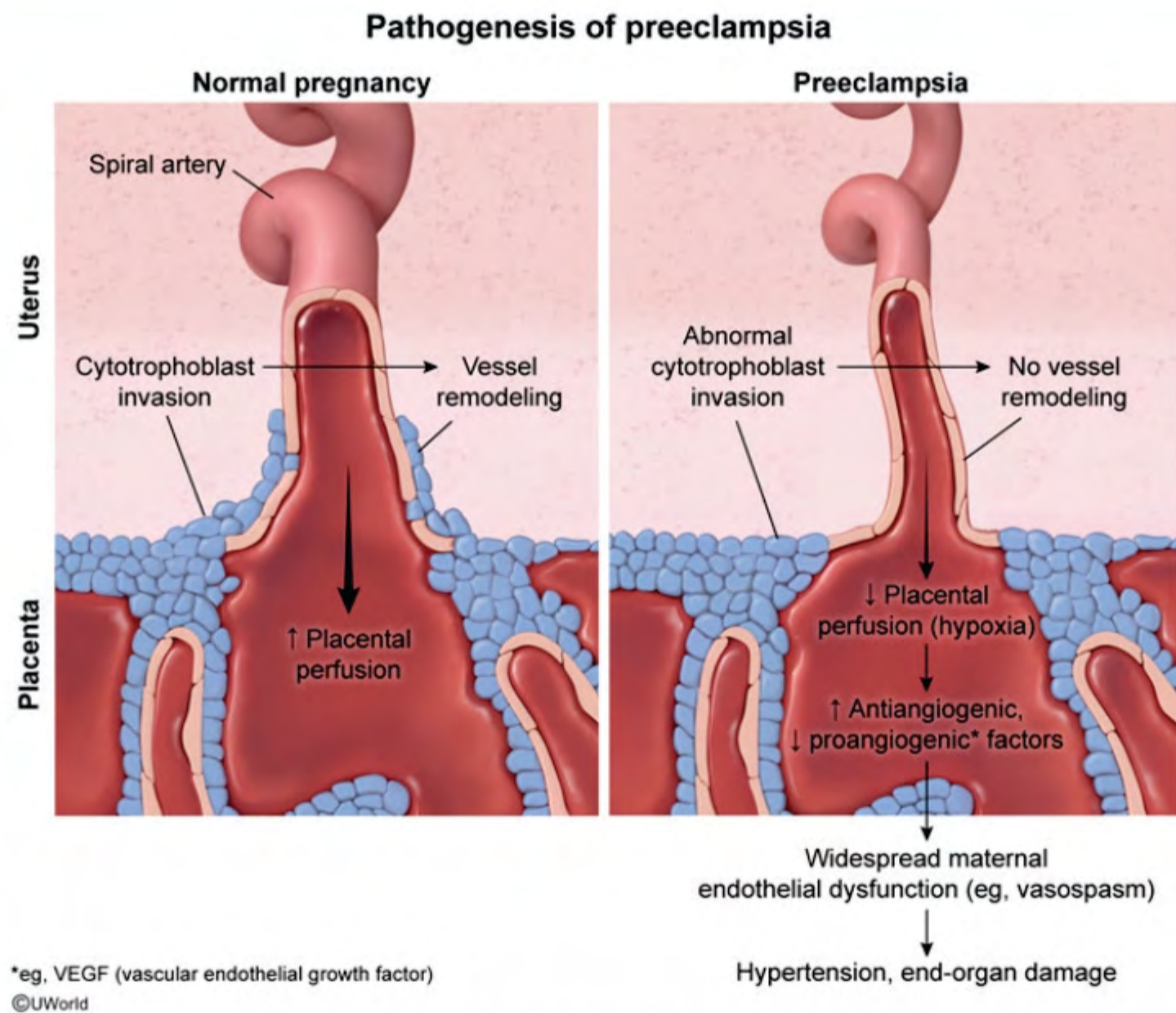
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Explanation



☐ D. Inflammatory optic nerve demyelination (5%)

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This patient has **preeclampsia**, defined as new-onset hypertension (systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg) at ≥ 20 weeks gestation with proteinuria and/or signs of end-organ dysfunction. Preeclampsia likely occurs due to abnormal placental development that results in high-resistance, low-perfusion vessels and subsequent placental ischemia. Placental ischemia triggers **widespread endothelial dysfunction**, which causes dysregulated vascular tone (eg, **vasospasm**, vasoconstriction), increased vascular permeability, and decreased end-organ perfusion.

Therefore, a classic presentation of preeclampsia is hypertension with **headache** and **visual changes**:

- Headaches are typically severe and throbbing in nature. They occur due to dysregulated cerebral blood flow, brain ischemia, and/or capillary leakage resulting in cerebral edema.
- Visual changes include blurry vision, photopsia (seeing "sparks"), and **scotomata** (partial vision loss [blind spots]), which are due to **retinal artery vasospasm** and optic nerve ischemia. Eye movement and the pupillary light reflex typically remain intact.

Symptoms usually resolve after delivery, which is curative for preeclampsia. However, in rare cases, patients with severe retinal ischemia or retinal detachment may have permanent vision loss.

(Choice A) Choroidal neovascularization, abnormal vessel growth beneath the retina, causes age-related wet macular degeneration. However, it presents with painless vision loss (no associated headache) and onset of symptoms is typically unilateral (ie, one eye affected first).

(Choice B) A decreased anterior chamber angle (ie, angle-closure glaucoma) can cause headache and visual changes due to increased intraocular pressure. However, patients typically have a painful red eye and a dilated pupil that reacts poorly to light.

(Choice C) Vitreous hemorrhage is associated with eye injury, anticoagulation, and diabetic retinopathy. It typically causes monocular vision loss associated with floaters/photopsia, not bilateral scotomata.

(Choice D) Inflammatory optic nerve demyelination (ie, optic neuritis) causes monocular vision loss and eye pain with extraocular movement. This patient has bilateral visual changes and normal extraocular movement.

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- Headaches are typically severe and throbbing in nature. They occur due to dysregulated cerebral blood flow, brain ischemia, and/or capillary leakage resulting in cerebral edema.
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Educational objective:

Preeclampsia causes widespread endothelial dysfunction, resulting in dysregulated vascular tone, increased vascular permeability, and decreased end-organ perfusion. Therefore, a common presentation is headache with visual changes (eg, scotomata) due to dysregulated cerebral blood flow and retinal artery vasospasm.

A 24-year-old woman comes to the clinic due to a missed menstrual period. She has had regular menstrual cycles every 28 days, and her last menstrual period began 7 weeks ago. Since then, she had sexual intercourse once 5 weeks ago. The patient has no chronic medical conditions. Urine pregnancy test is positive. Which of the following embryologic events is most likely occurring in the embryo at this time?

- ☐ A. Blastocyst implantation
- ☐ B. Gastrulation
- ☐ C. Intestinal fixation to abdominal wall
- ☐ D. Neural crest migration
- ☐ E. Primitive alveoli formation

Submit

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- A. Blastocyst implantation (8%)

✖

B. Gastrulation (22%)

C. Intestinal fixation to abdominal wall (6%)

✔

D. Neural crest migration (50%)

E. Primitive alveoli formation (11%)
- Incorrect

Correct answer D

50%

Answered correctly

12 secs













Time Spent

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Explanation

Fetal development*

Pre-organogenesis		Embryonic stage (weeks)						Fetal stage (weeks)			
1	2	3	4	5	6	7	8	9	16	20–36	38
											

* Adapted from Moore, Clinically Oriented Embryology, 10th ed, Figure 1.1

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By convention, gestational age is calculated from the first day of the last menstrual period, which was 7 weeks ago in this patient. However, **ovulation** typically occurs **2 weeks after a menstrual period** (and coincides with her sexual history), marking the approximate time that fertilization (conception) occurred. Early embryonic development is described in weeks postfertilization, and, in this patient, the embryo is estimated to be **5 weeks**.

Critical postfertilization stages include the following:

- **Implantation**: The blastocyst implants in the uterine wall at the end of week 1 postfertilization (**Choice A**).
- **Gastrulation**: After implantation, gastrulation occurs, which is the process that establishes 3 distinct cell layers (ectoderm, mesoderm, endoderm) from which all tissues and organs are derived. This process occurs during week 3 postfertilization (**Choice B**).
- **Neurulation**: Formation of the neural tube (precursor to the brain and spinal cord) occurs as the lateral edges of the neural plate (ie, neural folds) migrate toward the midline and fuse by the end of week 4 postfertilization. During this process, neuroectodermal cells adjacent to the crest of the neural folds separate from the surface ectoderm to form neural crest cells.

Following neurulation and by the end of **week 5 postfertilization**, these **neural crest cells migrate to distant sites** and later develop into diverse tissues (eg, neural ganglia, melanocytes, adrenal medulla). Based on this patient's menstrual and sexual history consistent with an embryo of 5 weeks, neural crest migration is likely occurring.

(Choice C) Rapid expansion of the gastrointestinal tract with physiologic herniation of the intestinal loops occurs during the early embryonic period (typically weeks 4-8) and is likely happening in this embryo. However, fixation of the intestines to the abdominal wall occurs after their return to the abdominal cavity (around weeks 10-12).

(Choice E) Although the primary bronchial buds develop during the embryonic stage (weeks 4-5), primitive alveoli form during the saccular stage of **lung development**, beginning at approximately week 26; alveoli continue to mature throughout gestation and after birth.

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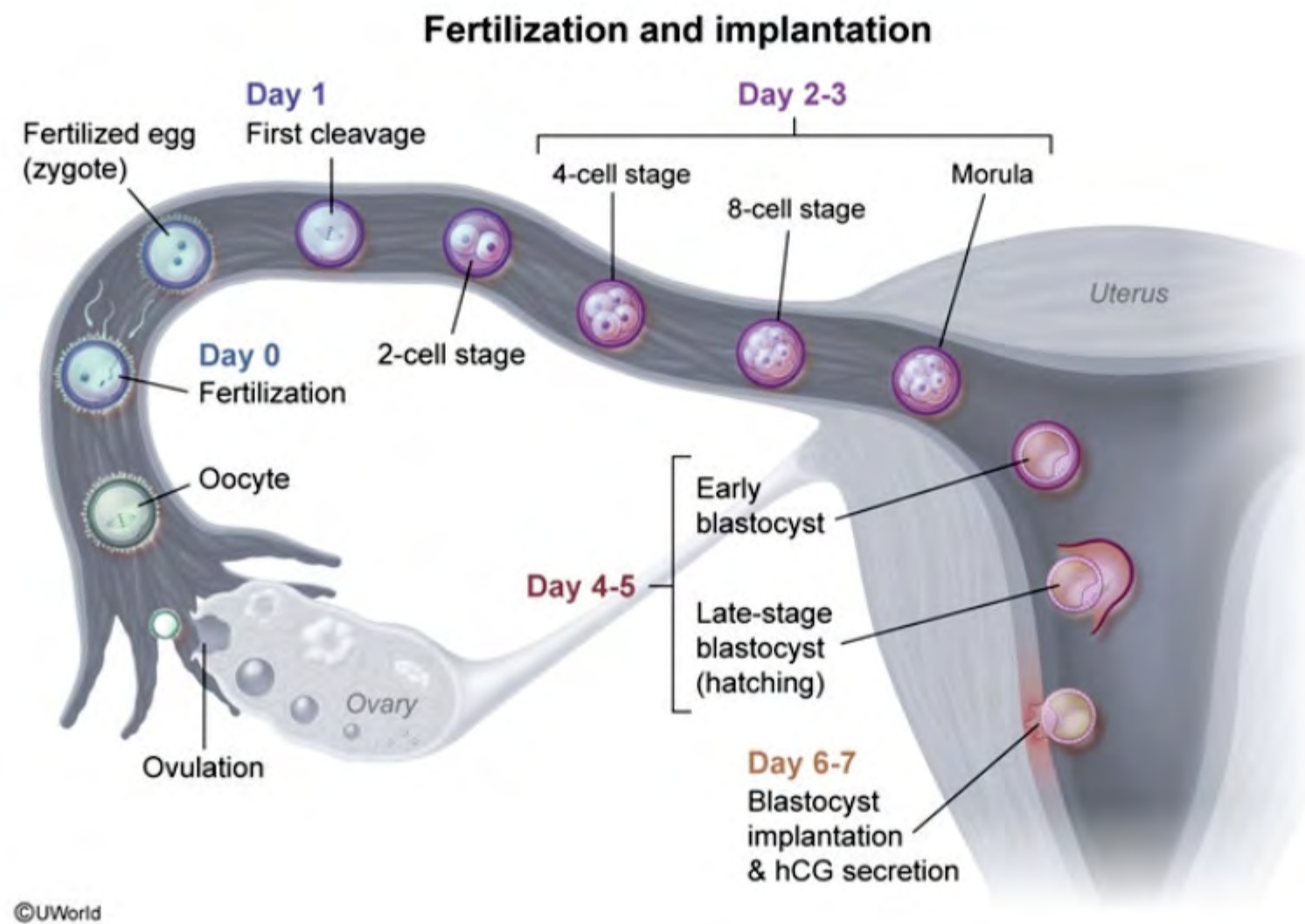
(Choice E) Although the primary bronchial buds develop during the embryonic stage (weeks 4-5), primitive alveoli form during the saccular stage of **lung development**, beginning at approximately week 26; alveoli continue to mature throughout gestation and after birth.

Educational objective:

Early embryonic development is described in weeks postfertilization (ie, approximate time of ovulation), and major events during this time include implantation (week 1), gastrulation (week 3), and neurulation (week 4). As neurulation occurs, neuroectodermal cells differentiate into neural crest cells, which then migrate to distant sites during week 5 postfertilization.

Palate

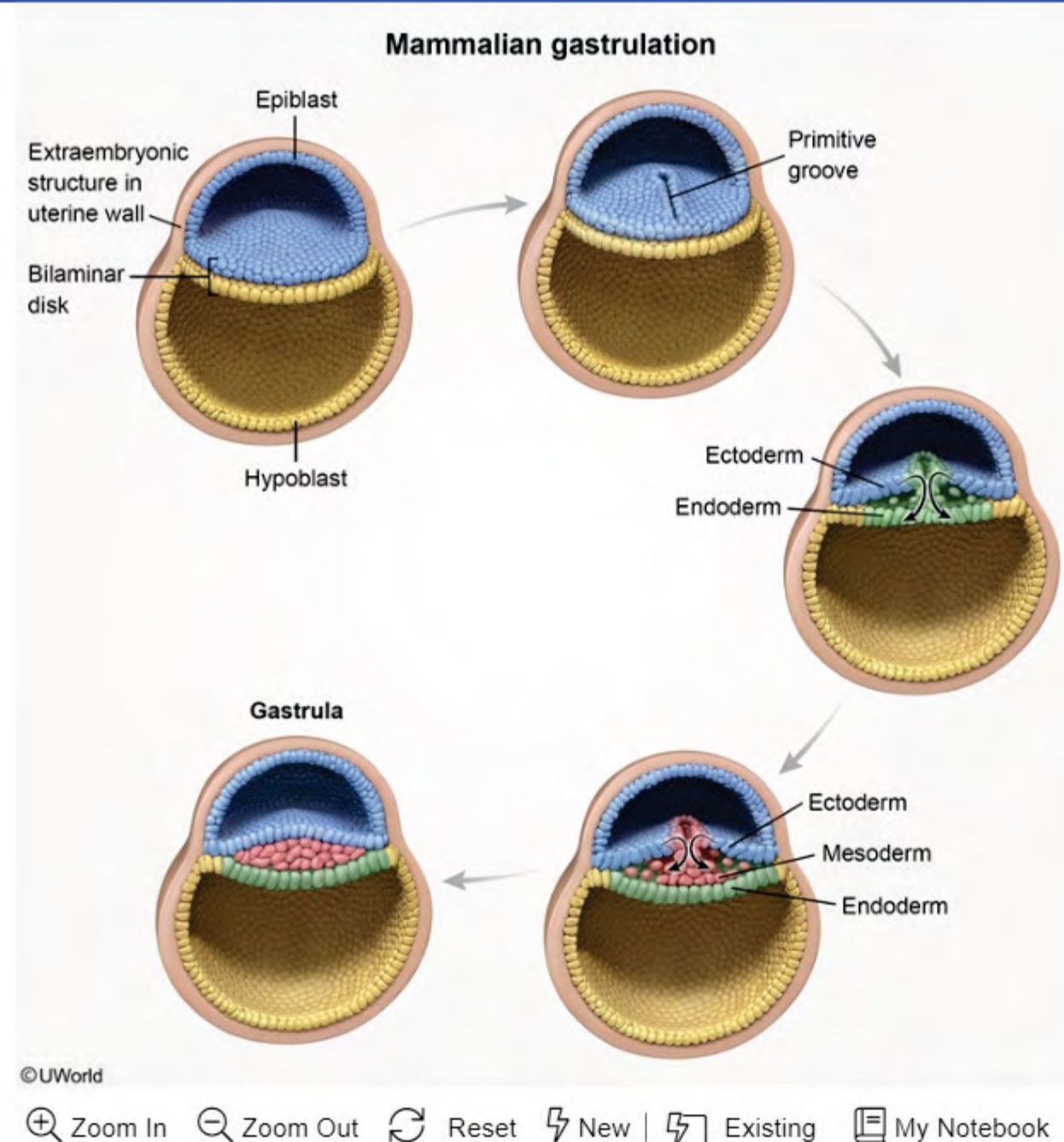
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Palate

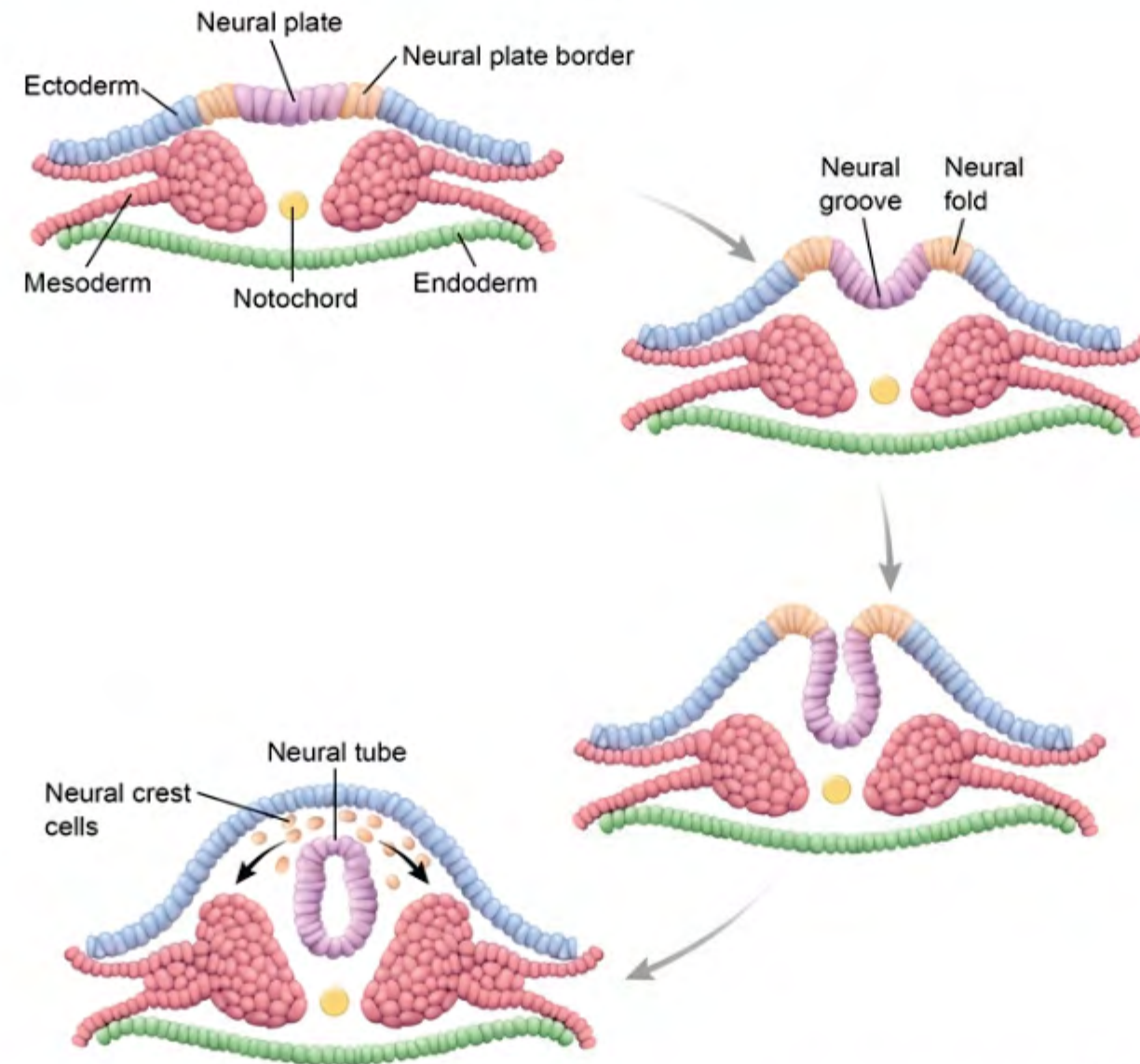
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Palate

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Neurulation



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• **Implantation:** The blastocyst implants in the uterine wall at the end of week 1 postfertilization (**Choice A**).

• **Embryonic development:** The embryo develops from the blastocyst into the fetus during the first trimester.

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Stages of lung development

Embryonic (3 - 5 weeks)	Pseudoglandular (5 - 16 weeks)	Canalicular (16 - 26 weeks)	Saccular (26 weeks - birth)	Alveolar (36 weeks - 8 years)
				
<ul style="list-style-type: none">• Lung bud forms trachea & bronchi	<ul style="list-style-type: none">• Endodermal tubules differentiate into terminal bronchioles	<ul style="list-style-type: none">• Respiratory bronchioles develop• Capillarization occurs	<ul style="list-style-type: none">• Alveolar ducts form terminal sacs• Pneumocytes begin producing surfactant	<ul style="list-style-type: none">• Alveoli undergo septation & proliferation

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A newborn boy is evaluated in the nursery. The patient was born at 38 weeks gestation via cesarean delivery due to breech presentation. The pregnancy was complicated by uterine fibroids. Head circumference, weight, and length are in the 75th to 90th percentiles. The neck is supple. Cardiopulmonary examination is unremarkable, and the abdomen is soft. The back appears unremarkable. Hip examination shows no signs of dislocation. There is excessive plantar flexion of both feet, with the forefeet pointing medially and the soles facing inward. There is full range of motion at the ankles; both feet can correct to the normal position. Muscle tone is normal and newborn reflexes are intact. The abnormal findings on this patient's physical examination most likely represent which of the following types of congenital anomalies?

- ☐ A. Deformation

☐ B. Disruption

☐ C. Dysplasia

☐ D. Malformation

☐ E. Sequence

Submit

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- ✓

☐

A. Deformation (52%)
- ✗

☒

B. Disruption (17%)
- ☐

C. Dysplasia (4%)
- ☐

D. Malformation (13%)
- ☐

E. Sequence (11%)

Incorrect

Correct answer
A

52%
Answered correctly

05 secs
Time Spent

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Explanation

Types of congenital anomalies		
Type	Definition	Example

Types of congenital anomalies		
Type	Definition	Example
Deformation	<ul style="list-style-type: none">Mechanical forces alter shape/position of a structure that was previously developing normally	<ul style="list-style-type: none">Clubfoot
Disruption	<ul style="list-style-type: none">Destruction of a structure that was previously developing normally	<ul style="list-style-type: none">Amniotic band
Dysplasia	<ul style="list-style-type: none">Abnormal organization of cells within a tissue (often genetic)	<ul style="list-style-type: none">Skeletal dysplasias (eg, achondroplasia)
Malformation	<ul style="list-style-type: none">Defect in organogenesis (eg, genetic, teratogenic)	<ul style="list-style-type: none">Spina bifidaHoloprosencephaly

This newborn patient has **talipes equinovarus** ([clubfoot](#)), a congenital anomaly in which one or both feet are excessively plantarflexed and swung inward. Clubfoot is a **deformation**, a type of anomaly that occurs when **abnormal mechanical forces** impinge upon an otherwise normally developing fetal structure, leading to **alterations in shape or position**. Risk factors include any condition that **restricts fetal movement**, such as breech presentation, uterine fibroids, or multiple gestation.

Although some deformations can improve once the extrinsic constraint is no longer present (ie, after birth), prolonged/severe mechanical forces in utero may cause significant, often permanent, structural changes. In cases of clubfoot, mechanical forces may contribute to defects in the developing bones, muscles, or tendons, resulting in a rigid abnormality (not seen in this patient with normal range of motion).

(Choice B) Disruption refers to arrested development or destruction of a structure that was previously developing normally; it is often triggered by interruption of the blood supply. For example, [amniotic bands](#), which are fibrous tissues that can encircle and constrict fetal limbs, may lead to amputation of a distal extremity. This patient's

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(Choice C) Dysplasia describes an inherent defect in tissue development due to the abnormal organization of cells. For example, achondroplasia is characterized by defective endochondral bone formation due to a genetic mutation resulting in skeletal anomalies.

(Choice D) A malformation is due to a primary defect (eg, genetic, teratogenic) in embryonic development that prevents the proper development of a structure. For example, **spina bifida**, a malformation of the spine and spinal cord, is caused by failure of neural tube closure (in contrast to clubfoot, in which limb development proceeds normally until late gestation)

(Choice E) A sequence refers to a group of anomalies that represent a cascade effect from a single defect. For instance, **Potter sequence** is characterized by flat facies, pulmonary hypoplasia, and limb deformities (eg, clubfoot) resulting from oligohydramnios due to a urinary tract anomaly. However, this patient has isolated feet deformities.

breech presentation, uterine fibroids, or multiple gestation.

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Educational objective:

A deformation anomaly is caused by abnormal extrinsic forces on a developing fetus. Talipes equinovarus (clubfoot) is an anomaly in which the foot is plantarflexed and pointed inward due to restricted in utero movement.

- Abnormal organization of cells within a tissue (often
- Skeletal dysplasias (eg.

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Positional clubfoot



Flexible
(can be passively repositioned
to normal positioning)

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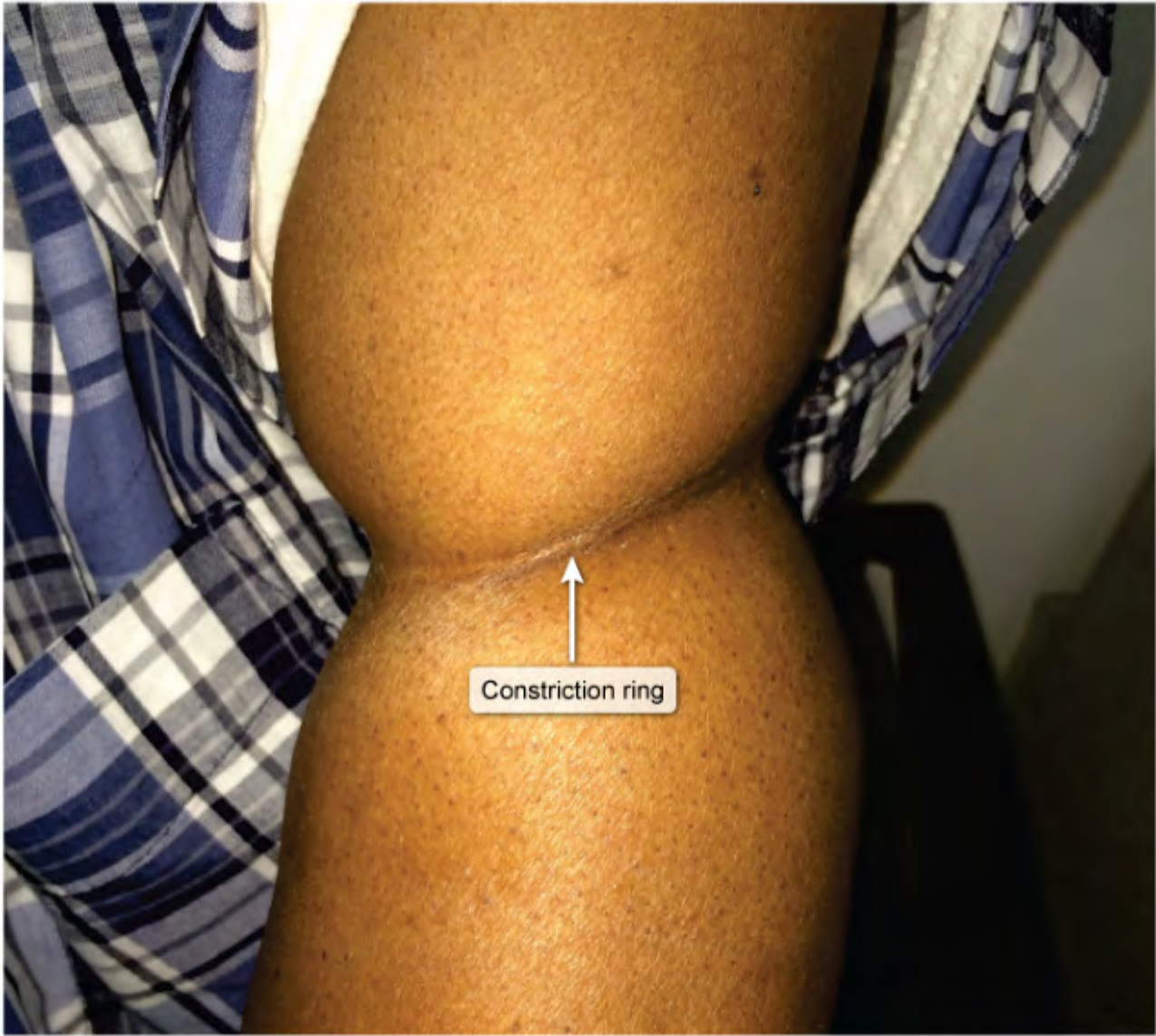
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Exhibit Display

Amniotic band syndrome



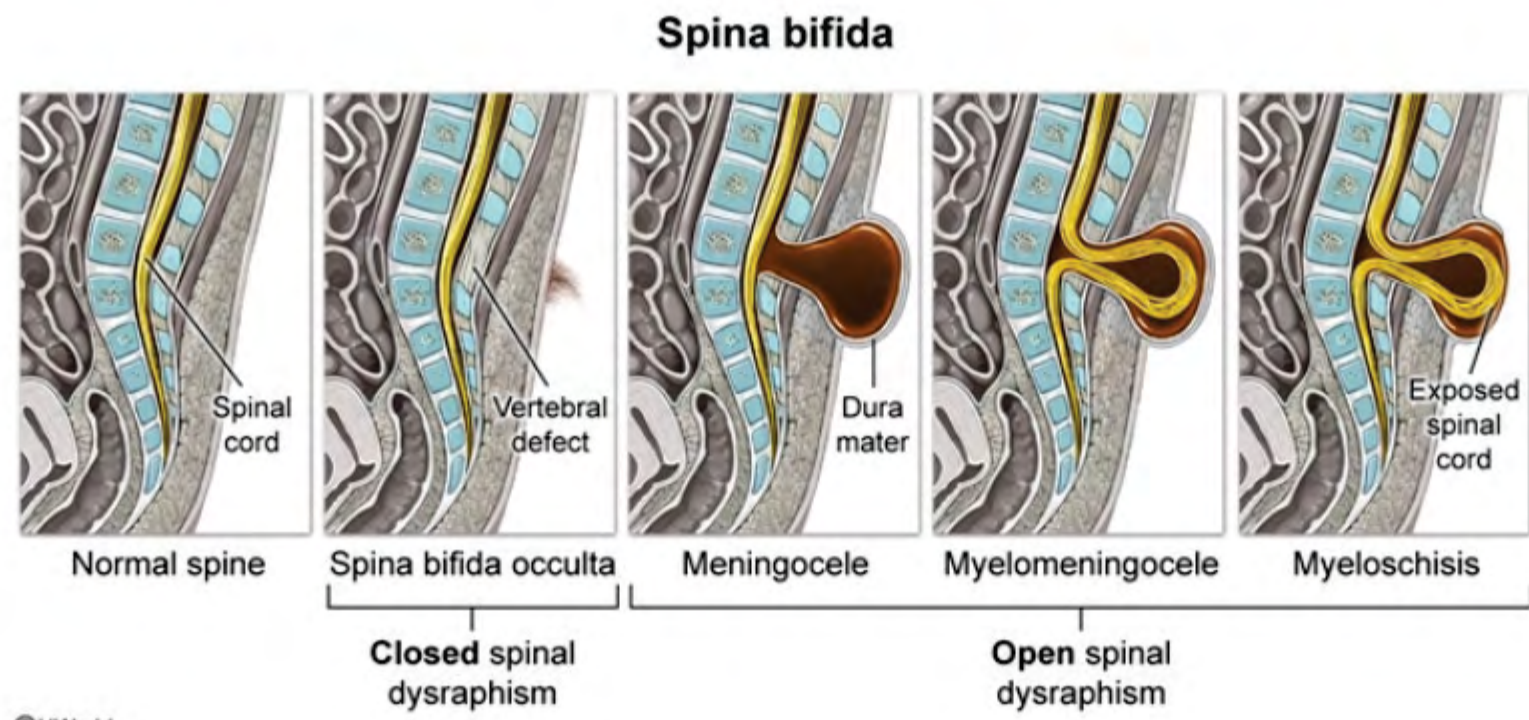
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breech presentation, uterine fibroids, or multiple gestation.

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(clubfoot) is an anomaly in which the foot is plantarflexed and pointed inward due to restricted in utero movement.

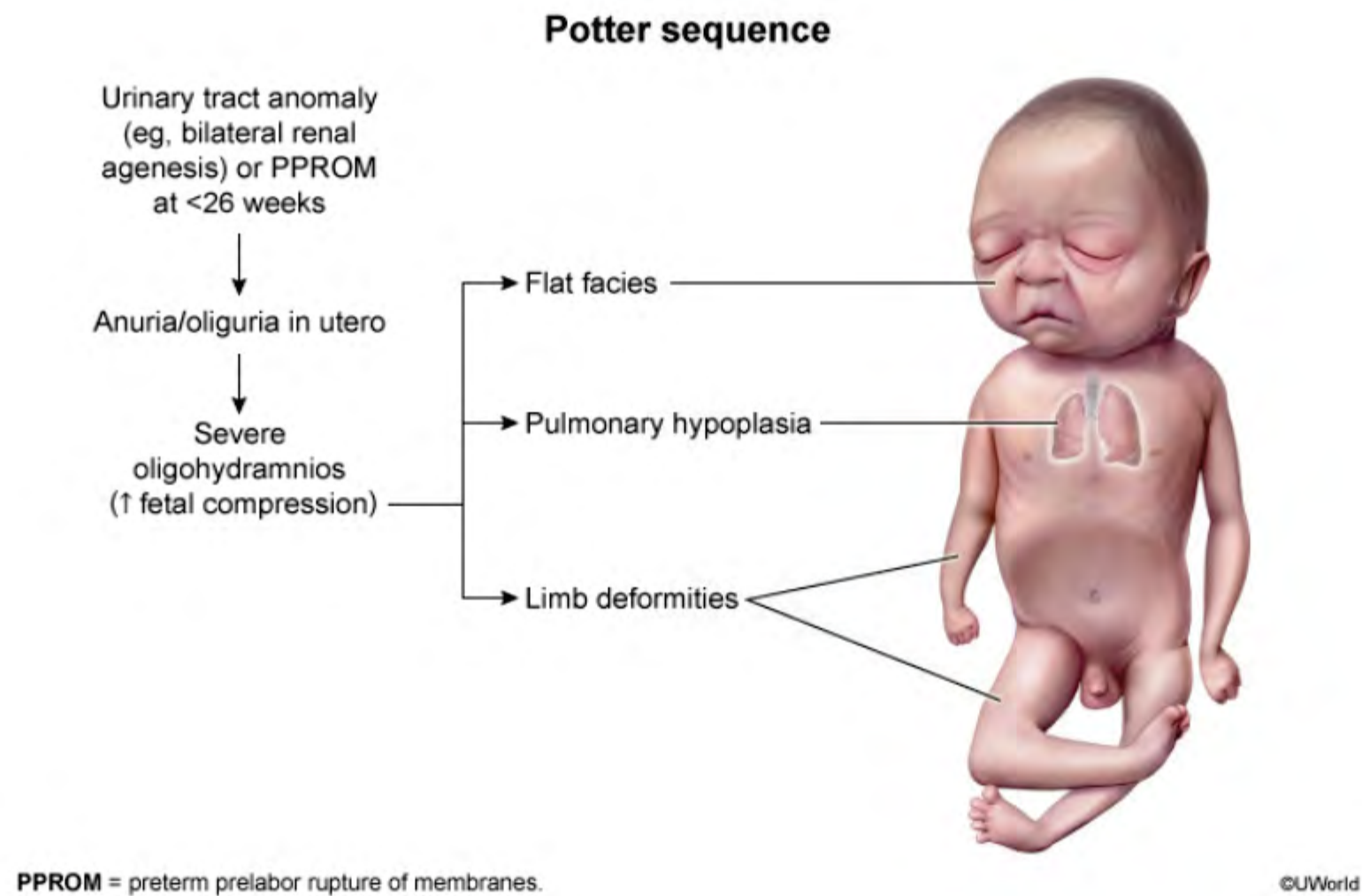
breech presentation, uterine fibroids, or multiple gestation.

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(clubfoot) is an anomaly in which the foot is plantarflexed and pointed inward due to restricted in utero movement.



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A 32-year-old woman comes to the office at 28 weeks gestation due to dyspnea. She has been feeling short of breath when she is supine but has no other symptoms. The patient has had no prenatal care during her pregnancy. She has a history of epilepsy that is well-controlled with medication. She lives with her husband and does not use tobacco, alcohol, or illicit drugs. Her immunizations are up to date, and she has no allergies. Physical examination shows a uterine size that is larger than expected for gestational age. Sonographic assessment shows markedly elevated amniotic fluid levels. Which of the following fetal anomalies would most likely account for this patient's polyhydramnios?

- ☐ A. Anencephaly
- ☐ B. Atrial septal defect
- ☐ C. Posterior urethral valves
- ☐ D. Pulmonary hypoplasia
- ☐ E. Renal agenesis
- ☐ F. Spina bifida occulta

Submit

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- ✓

☐ A. Anencephaly (58%)
- ✗

☒ B. Atrial septal defect (1%)
- ☐ C. Posterior urethral valves (5%)
- ☐ D. Pulmonary hypoplasia (14%)
- ☐ E. Renal agenesis (11%)
- ☐ F. Spina bifida occulta (8%)

Incorrect

Correct answer
A

58%

Answered correctly

06 secs

Time Spent

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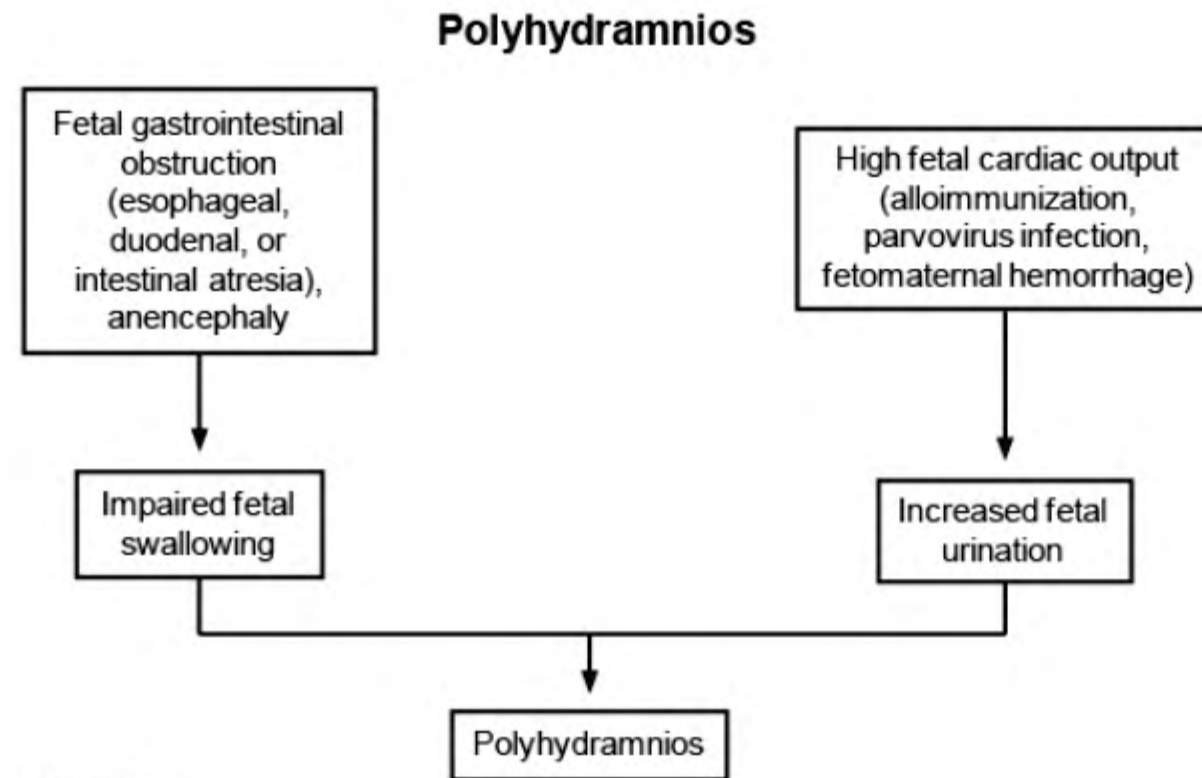
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Explanation



☐ D. Pulmonary hypoplasia (14%)

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Poly

uterine enlargement (eg, increased abdominal circumference) out of proportion to gestational age. Potential complications include preterm labor, placental abruption, and uterine atony due to uterine overdistention.

Polyhydramnios is the excessive accumulation of amniotic fluid. Moderate-to-severe polyhydramnios causes uterine enlargement (eg, increased abdominal circumference) out of proportion to gestational age. Potential complications include preterm labor, placental abruption, and uterine atony due to uterine overdistention. Polyhydramnios also increases the risk of **maternal respiratory compromise** as the abdominal cavity impairs lung expansion.

Polyhydramnios can be due to decreased fetal swallowing or increased fetal urination. Fetal anomalies associated with **impaired swallowing** include **gastrointestinal obstruction** (eg, duodenal, esophageal, or intestinal atresia) and **anencephaly** (a defect of the cranial neural tube). Causes of **increased fetal urination** include high cardiac output due to anemia or twin-to-twin transfusion syndrome. **Maternal diabetes** and **multiple gestations** tend to cause milder polyhydramnios compared to the aforementioned major fetal anomalies.

This patient's prenatal use of anti-epileptic therapy (eg, valproate, carbamazepine, phenytoin) is a substantial risk factor for fetal neural tube defects such as anencephaly. Like anencephaly, spina bifida (**Choice F**) is a group of neural tube defects (eg, occulta, meningocele, myelomeningocele) that can result from maternal folic acid deficiency and use of folic acid antagonists (anti-epileptic drugs, trimethoprim). However, spina bifida occulta, the mildest form, is not associated with polyhydramnios as fetal swallowing of amniotic fluid remains normal.

(Choice B) Atrial septal defects (ASDs) are one of the most common congenital heart diseases. ASDs are usually asymptomatic in utero. The diagnosis is usually made after birth based on wide fixed splitting of S2 on physical examination. ASDs are not associated with polyhydramnios.

(Choices C, D, and E) Renal agenesis is associated with severe oligohydramnios (too little amniotic fluid) after 16 weeks of gestation when the majority of the amniotic fluid is derived from fetal urine. **Posterior urethral valves** in male fetuses can also result in decreased fetal urine output and oligohydramnios. Oligohydramnios, in turn, can lead to excessive fetal compression causing flat facies and limb deformity (ie, **Potter sequence**). Pulmonary hypoplasia is a consequence of oligohydramnios and is not associated with polyhydramnios.

lung expansion.

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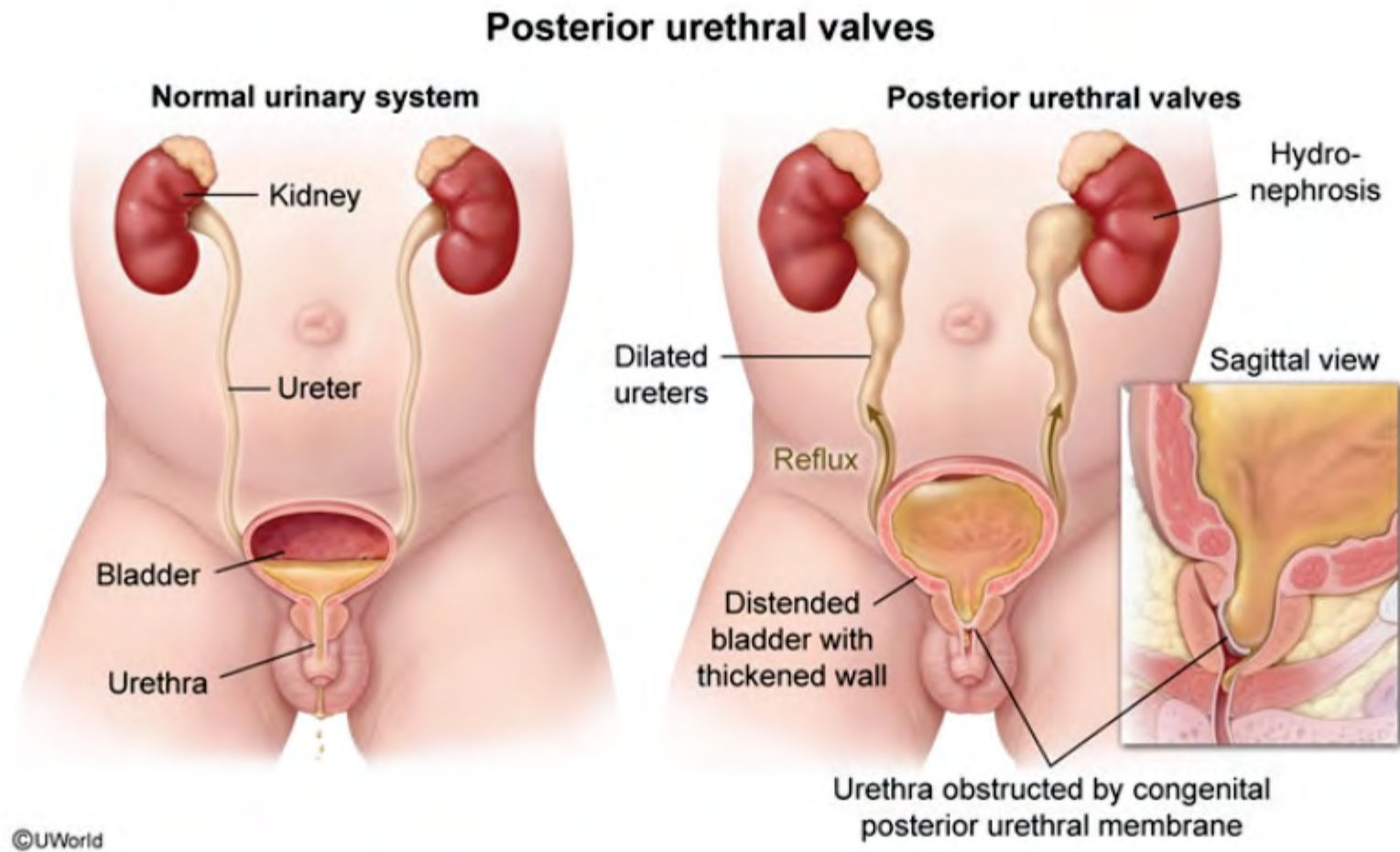
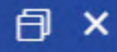
Educational objective:

Polyhydramnios (excessive accumulation of amniotic fluid) presents with increased abdominal circumference out of proportion to gestational age. The etiology is decreased fetal swallowing or increased fetal urination. Fetal anomalies associated with impaired swallowing include gastrointestinal obstruction (eg, duodenal, esophageal, or intestinal atresia) and anencephaly.

Polyhydramnios

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hypoplasia is a consequence of oligohydramnios and is not associated with polyhydramnios.

Polyhydramnios can be due to decreased fetal swallowing or increased fetal urination. Fetal anomalies associated with

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intestinal atresia) and anencephaly.

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Potter sequence

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graph TD; A["Urinary tract anomaly  
(eg, bilateral renal  
agenesis) or PPROM  
at <26 weeks"] --> B["Anuria/oliguria in utero"]; B --> C["Severe  
oligohydramnios  
(↑ fetal compression)"]; C --> D["Flat facies"]; C --> E["Pulmonary hypoplasia"]; C --> F["Limb deformities"];
```

PPROM = preterm prelabor rupture of membranes.

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A 21-day-old boy is brought to the office by his mother because of a palpable bulge in the child's neck. He continues to feed well but appears comfortable only when held with his body sideways under the breast. He is at the 50th percentile for height, weight, and head circumference. The child favors looking toward the right and cries when his head is turned to the left. There is a firm mass on the left side of his neck that does not move when the child swallows. The remainder of the examination is unremarkable. Which of the following conditions was most likely present prenatally?

- ☐ A. Folate deficiency
- ☐ B. Intrauterine malposition
- ☐ C. Defective fetal collagen synthesis
- ☐ D. Maternal alcohol consumption
- ☐ E. Upper respiratory infection in the mother

Submit

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- ☐

A. Folate deficiency (12%)
- ☒

B. Intrauterine malposition (47%)
- ☐

C. Defective fetal collagen synthesis (15%)
- ☐

D. Maternal alcohol consumption (9%)
- ☐

E. Upper respiratory infection in the mother (13%)

Correct

47%

Answered correctly

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Time Spent

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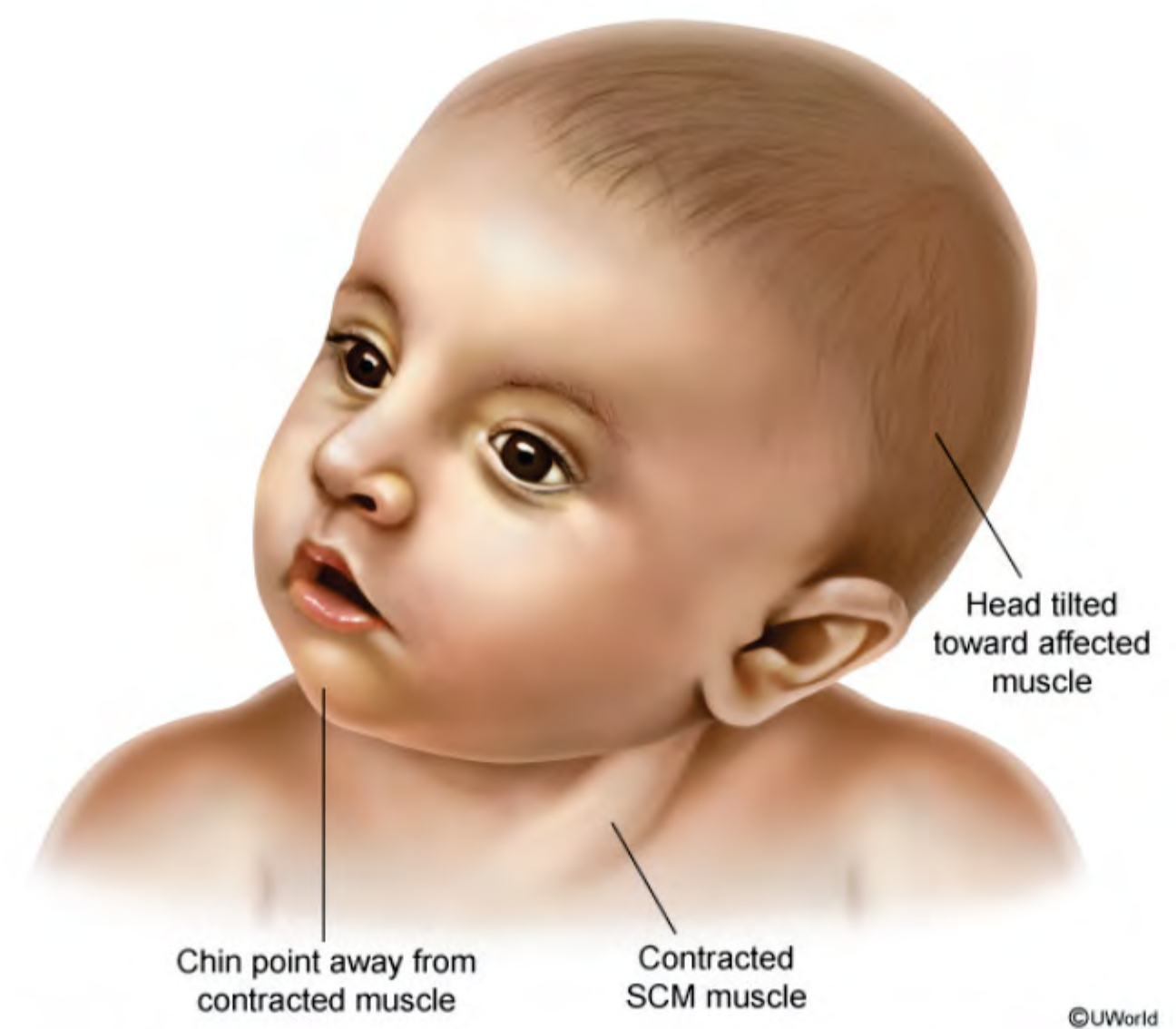
Version

Explanation



Exhibit Display

Congenital torticollis



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This infant has **congenital torticollis**, which typically develops by 2 to 4 weeks of age. It is most commonly caused by **birth trauma** (eg, breech delivery) or **malposition** of the head in utero (eg, due to fetal macrosomia or oligohydramnios), both of which can result in **sternocleidomastoid muscle (SCM) injury** and fibrosis. Children with congenital torticollis may have additional musculoskeletal anomalies, including hip dysplasia, metatarsus adductus (ie, adduction of the forefoot), and talipes equinovarus (ie, clubfoot).

The diagnosis of congenital torticollis is made clinically. On physical examination, the **head is tilted toward** the affected side with the **chin pointed away** from the contracture. A soft-tissue **mass** may be palpable in the inferior one-third of the affected SCM. Plagiocephaly and facial asymmetry may be seen in severe cases. Most cases resolve with conservative therapy and stretching exercises.

(Choice A) Folate deficiency during the first trimester of pregnancy is associated with fetal neural tube defects (eg, spina bifida).

(Choice C) Defective collagen synthesis causes osteogenesis imperfecta (brittle bone disease). Patients with the lethal variety usually die in utero or in early infancy due to severe fractures, pulmonary failure, or intracerebral hemorrhage.

(Choice D) Alcohol consumption during pregnancy can cause fetal alcohol syndrome. Affected infants have poor growth, functional developmental deficits, and characteristic facial abnormalities including a smooth philtrum, thin vermilion border, and small palpebral fissures.

(Choice E) The majority of upper respiratory infections in pregnancy are benign. However, rubella, one of the TORCH infections, is transmitted by the respiratory route and can cause severe birth defects, including hearing loss, cataracts, and congenital heart defects.

Educational objective:

Congenital torticollis is typically noted by 2 to 4 weeks of age, after which the child prefers to hold the head tilted to one side. It is most commonly the result of malposition of the head in utero or birth trauma. Most cases resolve with conservative therapy and stretching exercises.

A 33-year-old woman, gravida 2 para 1, comes to the office due to a positive home urine pregnancy test. Her last menstrual period was 6 weeks ago, and pelvic ultrasonography confirms an intrauterine pregnancy. The patient has idiopathic pulmonary hypertension complicated by Eisenmenger syndrome. She is aware that pregnancy poses a major mortality risk to herself and the fetus due to the hemodynamic changes of gestation, labor, and delivery. The patient consents to pregnancy termination with a mifepristone and misoprostol regimen. Which of the following is the mechanism of action for mifepristone in this clinical situation?

- ☐ A. Cyclooxygenase inhibitor
- ☐ B. Folic acid antagonist
- ☐ C. Oxytocin receptor agonist
- ☐ D. Progesterone antagonist
- ☐ E. Prostaglandin agonist

Submit

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- ✖

B. Folic acid antagonist
- C. Oxytocin receptor agonist
- ✔

D. Progesterone antagonist
- E. Prostaglandin agonist

Incorrect

Correct answer
D

Collecting Statistics

04 secs
Time Spent

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Version

Explanation

Medications used for pregnancy termination	
Methotrexate	<ul style="list-style-type: none">Ectopic pregnancyFolic acid antagonist (inhibits dihydrofolate reductase)Preferentially destroys proliferating fetal cells

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Mifepristone	<ul style="list-style-type: none">AbortionPartial progesterone agonist (acts as progesterone antagonist during pregnancy)Promotes placental separation & uterine contractions
Misoprostol	<ul style="list-style-type: none">AbortionProstaglandin E1 agonistStimulates uterine contractions

In the second half of the menstrual cycle, progesterone stimulates the development of secretory endometrium to create an environment favorable for implantation. In the first 7 weeks of pregnancy, progesterone is secreted by the corpus luteum; thereafter, its production is assumed by the placenta. **Mifepristone** is a **progesterone antagonist** that binds progesterone receptors with greater affinity than the natural hormone. Progesterone receptor blockade results in apoptosis and **necrosis of the uterine decidua** and prevents further development of a first trimester pregnancy.

When misoprostol is used with mifepristone, pregnancy termination success rate is increased. **Misoprostol**, a **prostaglandin E1 analog (Choice E)**, causes cervical softening and uterine contractions leading to expulsion of the pregnancy.

(Choice A) Indomethacin (cyclooxygenase inhibitor) is a tocolytic drug used to stop preterm labor by inhibiting prostaglandin synthesis, which decreases uterine contractility.

(Choice B) Folic acid antagonists (eg, methotrexate) inhibit trophoblast division, therefore decreasing trophoblast

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(Choice A) Indomethacin (cyclooxygenase inhibitor) is a tocolytic drug used to stop preterm labor by inhibiting prostaglandin synthesis, which decreases uterine contractility.

(Choice B) Folic acid antagonists (eg, methotrexate) inhibit trophoblast division, therefore decreasing trophoblast survival, hindering implantation, and encouraging expulsion. Methotrexate is used for the treatment of ectopic pregnancy and for termination of intrauterine pregnancy in conjunction with misoprostol when mifepristone is not available.

(Choice C) Oxytocin is a peptide hormone released by the posterior pituitary that stimulates uterine contractions. Myometrial oxytocin receptor concentration increases at term; therefore, oxytocin has limited effects on the uterus in early gestation and does not cause pregnancy termination.

Educational objective:

Progesterone is necessary for implantation and maintenance of pregnancy. Mifepristone is a progesterone antagonist that is used with misoprostol (a prostaglandin-E1 agonist) to terminate a first-trimester pregnancy.

References

- Practice bulletin no. 143: medical management of first-trimester abortion.

A 22-year-old unvaccinated woman comes to the office due to a rash that developed during a recent trip to Southeast Asia. A week ago, while still abroad, she developed a low-grade fever and arthralgias, followed by numerous pinpoint, pink macules and papules on her face. Over the next 48 hours, the rash spread to her trunk and then her extremities. She went to a local emergency department, where she was diagnosed with a viral illness and also found to be approximately 8 weeks pregnant. Her symptoms resolved over the following few days, and she returned to the United States. Physical examination today reveals postauricular lymphadenopathy. Heart and lung examinations are unremarkable. Fetal heart tones are normal. The fetus is at highest risk for developing which of the following complications?

- ☐ A. Epicanthic folds and brachycephaly
- ☐ B. Hydrocephalus and chorioretinitis
- ☐ C. Meningitis and malformed teeth
- ☐ D. Microcephaly and hearing loss
- ☐ E. Mucocutaneous vesicles and keratitis

Submit

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- A. Epicanthic folds and brachycephaly (0%)
- ✗

B. Hydrocephalus and chorioretinitis (10%)
- C. Meningitis and malformed teeth (2%)
- ✓

D. Microcephaly and hearing loss (81%)
- E. Mucocutaneous vesicles and keratitis (5%)

Incorrect

Correct answer
D

81%
Answered correctly

03 secs
Time Spent

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Explanation

Key features of congenital infections*	
Toxoplasmosis	<ul style="list-style-type: none">ChorioretinitisHydrocephalus

Key features of congenital infections*	
Toxoplasmosis	<ul style="list-style-type: none">ChorioretinitisHydrocephalusDiffuse intracranial calcifications
Syphilis	<ul style="list-style-type: none">RhinorrheaSkeletal anomaliesDesquamating rash (palms/soles)
Rubella	<ul style="list-style-type: none">CataractsHeart defects (eg, PDA)Sensorineural hearing loss
Cytomegalovirus	<ul style="list-style-type: none">Periventricular calcificationsMicrocephalySensorineural hearing loss
Herpes simplex virus	<ul style="list-style-type: none">Vesicular/ulcerative rash
<p>*Nonspecific findings include growth restriction, jaundice, hepatosplenomegaly & blueberry muffin rash.</p> <p>PDA = patent ductus arteriosus.</p>	

This **unvaccinated** woman with a recent travel history developed a low-grade fever and a **maculopapular rash** with **cephalocaudal spread**, findings characteristic of measles or rubella. In this case, **rubella** is the most likely cause because polyarthritis/**polyarthralgia**, particularly in women, is a classic finding with acute infection. In addition, localized occipital and/or **postauricular lymphadenopathy** is more often seen in rubella.

Transplacental transmission of the rubella virus to the fetus during the first trimester causes congenital rubella syndrome. **Sensorineural hearing loss, cataracts,** and cardiac malformations (eg, **patent ductus arteriosus**)

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Transplacental transmission of the rubella virus to the fetus during the first trimester causes congenital rubella syndrome. **Sensorineural hearing loss**, **cataracts**, and cardiac malformations (eg, **patent ductus arteriosus**) are classic manifestations in the affected infant. **Growth restriction** in weight, length, and head circumference (ie, **microcephaly**) is also common due to diffuse fetal inflammation.

Although treatment of suspected rubella infection is supportive care alone, the diagnosis should be confirmed in patients at risk for serious sequelae, such as pregnant women or newborns with classic findings. The presence of rubella IgM antibodies is diagnostic.

(Choice A) Epicanthic folds and brachycephaly are typical features of **Down syndrome**, a genetic disorder caused by an extra copy of chromosome 21, not by maternal infection during pregnancy.

(Choice B) Hydrocephalus and chorioretinitis are seen with congenital toxoplasmosis. *Toxoplasma gondii* infection in pregnant women is usually asymptomatic, but a nonspecific flu-like illness (eg, fever) with a maculopapular rash and lymphadenopathy can occur. In contrast to the diffuse onset of rash in toxoplasmosis, this patient's cephalocaudal progression of rash is characteristic of rubella. In addition, muscle aches (not joint pain) and cervical (not postauricular) lymph node swelling can occur in toxoplasmosis.

(Choice C) Meningitis and **malformed teeth** are consistent with congenital syphilis infection. In adults, secondary syphilis can present with constitutional symptoms (eg, fever, arthralgias), lymphadenopathy, and rash. However, lymphadenopathy is typically generalized, and the rash is classically present on the palms and soles.

(Choice E) Mucocutaneous vesicles and keratitis are manifestations of congenital herpes simplex virus (HSV) infection. In adults with HSV, head and neck lymphadenopathy can occur, but both primary and recurrent disease are associated with vesicular not maculopapular lesions that typically involve the oropharynx. More over

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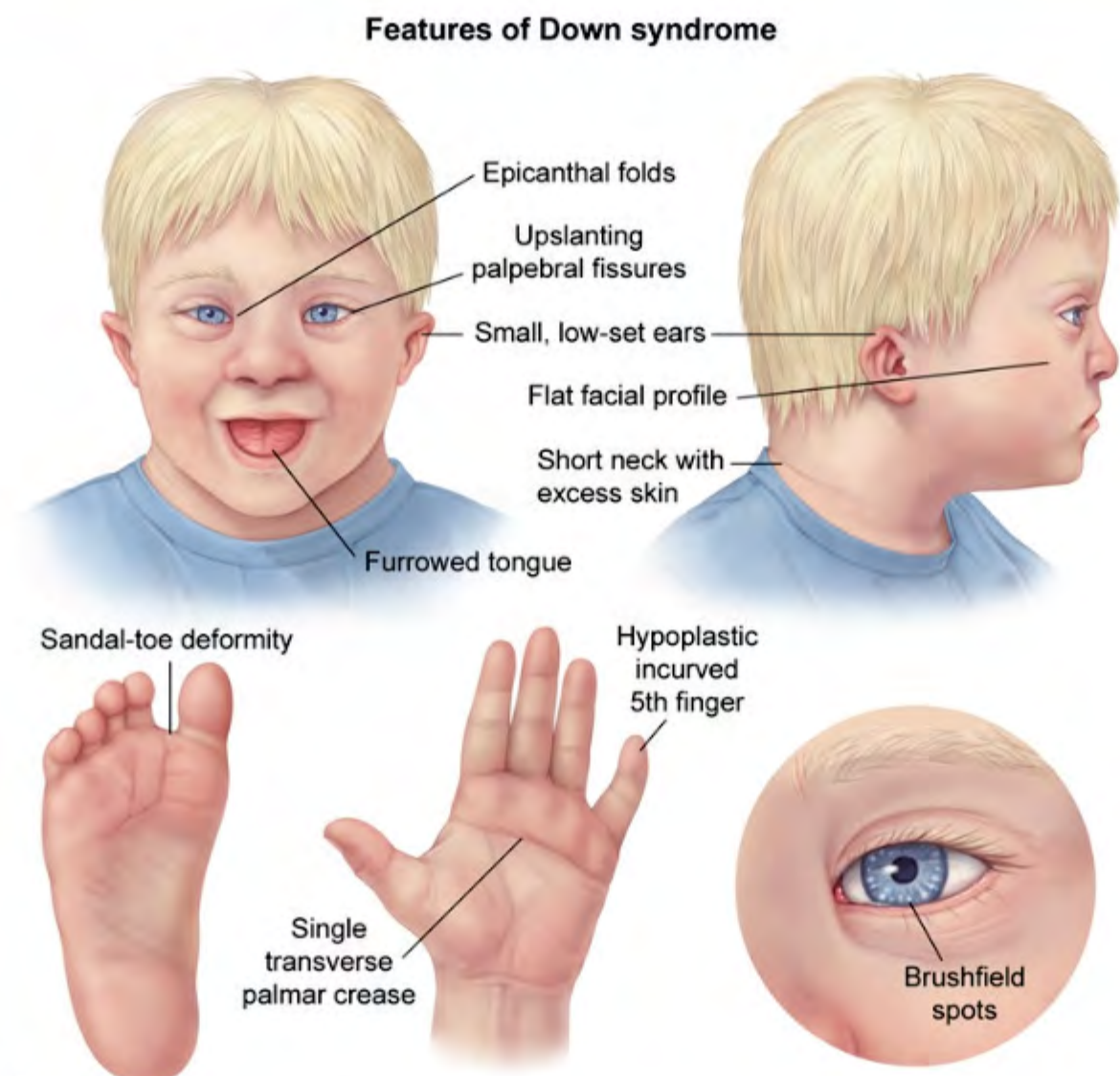
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Educational objective:

Maternal rubella infection classically causes a maculopapular rash with cephalocaudal progression, joint pain, and postauricular lymphadenopathy. Transplacental transmission to the fetus leads to congenital rubella syndrome, which is characterized by sensorineural deafness, cataracts, patent ductus arteriosus, and growth restriction (eg, microcephaly).

- Sensorineural hearing loss

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A 41-year-old woman, gravida 3 para 2, at 34 weeks gestation comes to the office for a routine prenatal visit. For the past 2 weeks, the patient has had increasing pain over the pubic symphysis that is unrelieved with acetaminophen or warm compresses. She has had no dysuria, hematuria, contractions, or vaginal bleeding. The patient has hypothyroidism that is well controlled with levothyroxine. Vital signs are normal. Examination shows a tender, slightly widened pubic symphysis. Sensation is intact. Which of the following hormones is the most likely underlying cause of this patient's pain?

- ☐ A. Growth hormone
- ☐ B. Human chorionic gonadotropin
- ☐ C. Insulin-like growth factor
- ☐ D. Oxytocin
- ☐ E. Relaxin

Submit

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- A. Growth hormone (5%)

✖

B. Human chorionic gonadotropin (9%)

C. Insulin-like growth factor (9%)

D. Oxytocin (31%)

✔

E. Relaxin (43%)

Incorrect

Correct answer
E

43%

Answered correctly

03 secs

Time Spent

2023

Version

Explanation

This patient has a slightly **widened pubic symphysis**, a common but often painful adaptation of pregnancy that occurs due to **increased relaxin levels**.

Relaxin is a hormone produced by the corpus luteum (in the first trimester) and the placenta (in the second and third trimesters). Its primary role during pregnancy is to promote physiologic changes to the maternal musculoskeletal system. Relaxin causes **increased ligament elasticity**, which is of particular importance in the

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Relaxin is a hormone produced by the corpus luteum (in the first trimester) and the placenta (in the second and third trimesters). Its primary role during pregnancy is to promote physiologic changes to the maternal musculoskeletal system. Relaxin causes **increased ligament elasticity**, which is of particular importance in the pubic symphysis and sacroiliac joints. The increased laxity of these pelvic joints increases pelvic mobility, widens the pelvic outlet, and changes the angle of the ilium to the sacrum – all of which **facilitate vaginal delivery**.

Although these musculoskeletal adaptations are physiologic, in some patients (particularly those who are multiparous or obese) there is excessive or asymmetric joint laxity, which leads to joint destabilization and pain over the **pubic symphysis** or the sacroiliac joints.

(Choices A and C) During pregnancy, growth hormone secretion by the maternal pituitary gland is gradually replaced by placental growth hormone (PGH) secreted by placental syncytiotrophoblasts. PGH directly stimulates placental development and promotes fetal growth by mobilizing maternal energy stores (eg, increasing gluconeogenesis, lipolysis).

(Choice B) Human chorionic gonadotropin (hCG) is secreted by placental syncytiotrophoblasts. The role of hCG is to signal the ovary to maintain the corpus luteum for continued progesterone production in early pregnancy.

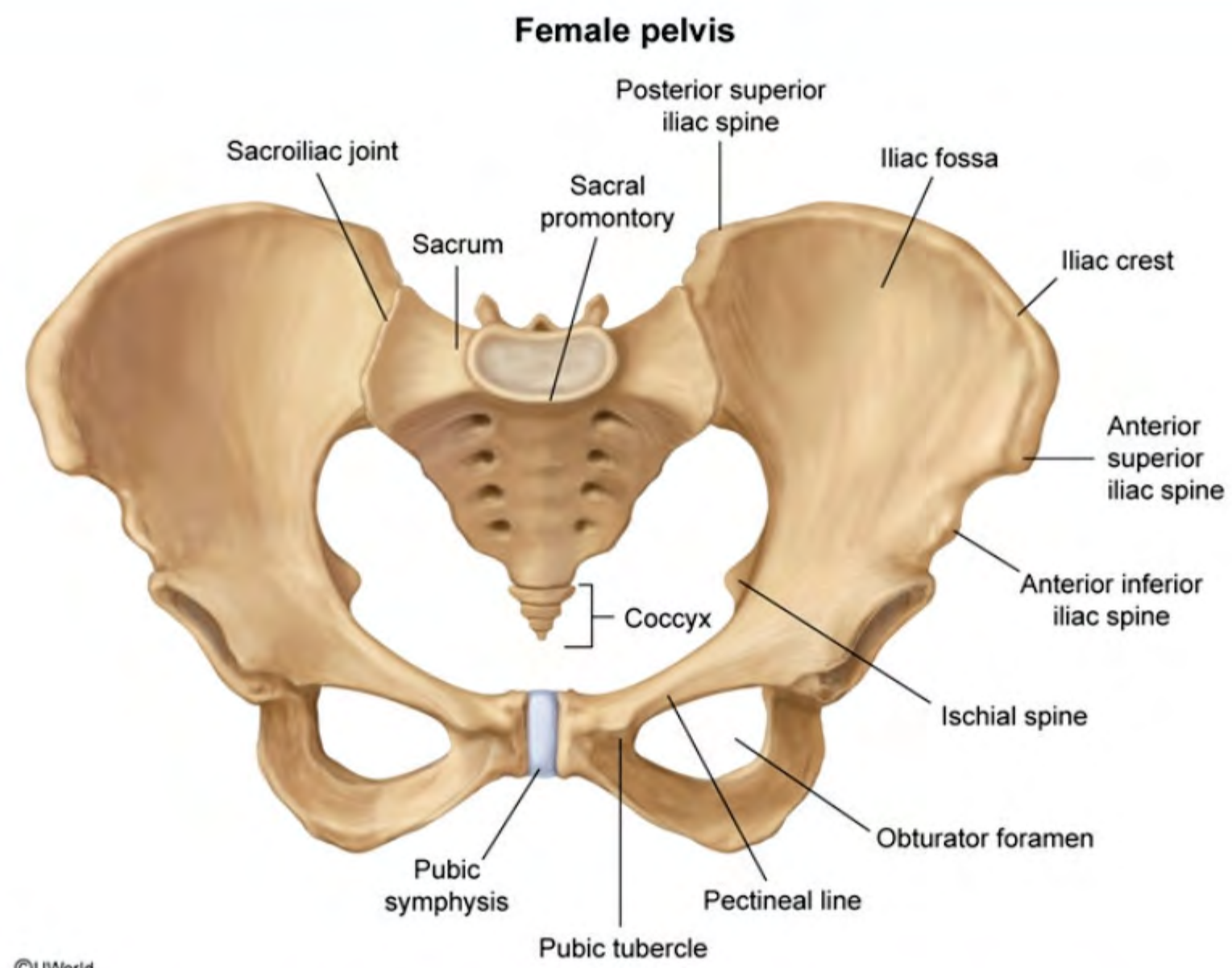
(Choice D) Oxytocin is a hormone released from the maternal pituitary gland; its primary role in pregnancy is to stimulate uterine contractions during labor.

Educational objective:

During pregnancy, increased relaxin levels promote sacroiliac joint laxity and widening of the pubic symphysis to help facilitate vaginal delivery.

References

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References

A 16-year-old girl comes to the emergency department with vaginal bleeding. Her last menstrual period was 12 weeks ago. She is sexually active and does not use contraception. The patient had a miscarriage last year that required a dilation and curettage. Urine pregnancy test is positive. Transvaginal ultrasound demonstrates an intrauterine gestational sac without fetal cardiac activity, and a dilation and curettage is performed. Pathology shows fetal tissue, focal trophoblastic hyperplasia, and some enlarged villi interspersed with normal villi. Which of the following is the most likely diagnosis?

- ☐ A. Choriocarcinoma
- ☐ B. Complete mole
- ☐ C. Invasive mole
- ☐ D. Miscarriage
- ☐ E. Partial mole
- ☐ F. Placental site trophoblastic tumor

Submit

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- A. Choriocarcinoma (2%)
- ✗

B. Complete mole (8%)
- C. Invasive mole (1%)
- D. Miscarriage (5%)
- ✓

E. Partial mole (81%)
- F. Placental site trophoblastic tumor (1%)

Incorrect

Correct answer
E

81%
Answered correctly

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Explanation

Histology of gestational trophoblastic disease				
Diagnosis	Classification	Trophoblasts	Villi	Fetal/embryonic tissue

Histology of gestational trophoblastic disease				
Diagnosis	Classification	Trophoblasts	Villi	Fetal/embryonic tissue
Partial mole	Benign	Focally hyperplastic	Focally enlarged, hydropic	Present, triploid
Complete mole	Benign	Diffusely hyperplastic	Diffusely enlarged, hydropic	Absent
Invasive mole	Malignant	Diffusely hyperplastic with myometrial invasion	Diffusely enlarged, hydropic	Absent
Gestational choriocarcinoma	Malignant	Diffusely anaplastic/necrotic with vascular invasion	Absent	Present or absent

A hydatidiform mole is a premalignant gestational trophoblastic disease (GTD) characterized by abnormal placental trophoblastic proliferation, resulting in **markedly elevated β -hCG** levels. Molar pregnancies are benign and result from aberrant fertilization and **overexpression of paternal genes**. Risk factors include extremes of maternal age, prior molar pregnancy, and prior miscarriage.

This patient has a **partial hydatidiform mole**, which typically presents in early pregnancy with **vaginal bleeding** and no fetal cardiac activity, similar to a miscarriage. Partial moles have a **triploid karyotype (69,XXX or XXY)** containing maternal and paternal DNA, with an extra chromosome set of paternal origin. Evacuated uterine contents contain **fetal tissue** and other parts (eg, cord, amniotic membrane), some edematous villi with **focal**

A hydatidiform mole is a premalignant gestational trophoblastic disease (GTD) characterized by abnormal placental trophoblastic proliferation, resulting in **markedly elevated β -hCG** levels. Molar pregnancies are benign and result from aberrant fertilization and **overexpression of paternal genes**. Risk factors include extremes of maternal age, prior molar pregnancy, and prior miscarriage.

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A complete mole (**Choice B**) may also present with vaginal bleeding. In addition, the uterus is enlarged out of proportion to the gestational age, and a **snowstorm pattern** is pathognomonic. Pre-eclampsia, hyperthyroidism, hyperemesis, and theca-lutein cysts may be present. Cells of the evacuated tissue have a 46,XX (or rarely 46,XY) karyotype with only paternal DNA. Histologically, no fetal tissue is present, and only edematous villi are seen.

(Choice A) Choriocarcinoma is a malignant GTD that can develop after any pregnancy. It often presents with vaginal bleeding and pulmonary metastases. Pathology shows sheets of anaplastic cytotrophoblasts and syncytiotrophoblasts and no villi.

(Choice C) An invasive mole is diagnosed by histologic evidence of villi invading the myometrium on a hysterectomy specimen. This can be the malignant progression of a complete mole.

(Choice D) A miscarriage is a pregnancy loss prior to 20 weeks gestation and presents with vaginal bleeding and low β -hCG level. Pathology would show fetal tissue and normal villi and trophoblastic tissue.

(Choice F) A placental site trophoblastic tumor is a proliferation of intermediate trophoblasts. This malignant type of GTD produces human placental lactogen.

Educational objective:

prior molar pregnancy, and prior miscarriage.

This patient has a **partial hydatidiform mole**, which typically presents in early pregnancy with **vaginal bleeding** and no fetal cardiac activity, similar to a miscarriage. Partial moles have a **triploid karyotype (69,XXX or XXY)** containing maternal and paternal DNA, with an extra chromosome set of paternal origin. Evacuated uterine contents contain **fetal tissue** and other parts (eg, cord, amniotic membrane), some edematous villi with **focal trophoblastic proliferation**, and some normal-appearing villi.

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Educational objective:

A partial mole will have a triploid karyotype (eg, 69,XXX or XXY) and contain fetal tissue with some edematous villi with focal trophoblastic proliferation, and normal-appearing villi. Patients present with vaginal bleeding, and prior miscarriage is a risk factor.

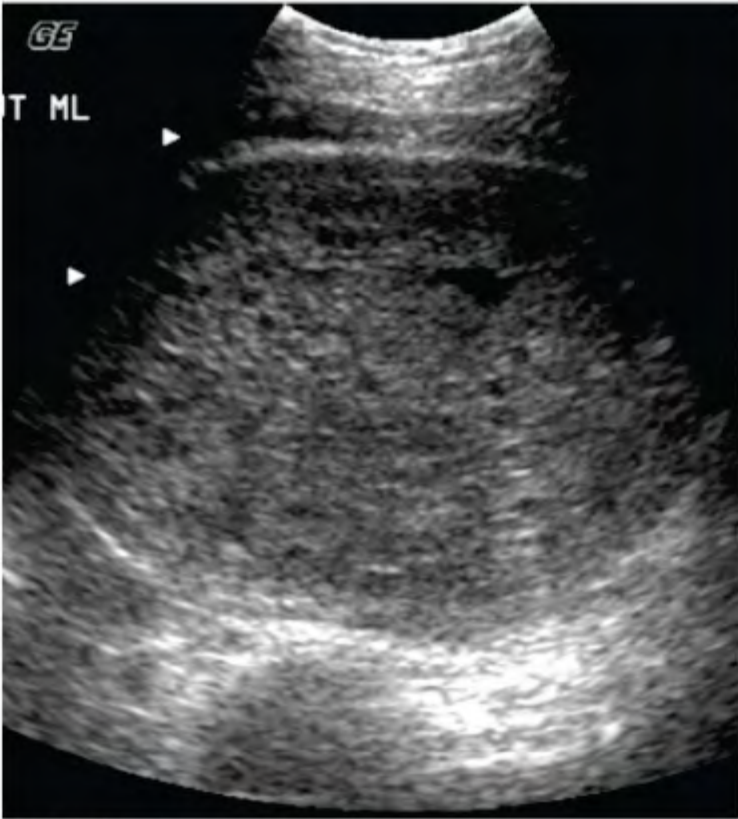
Reference

Gestational

Diffusely anaplastic/necrotic

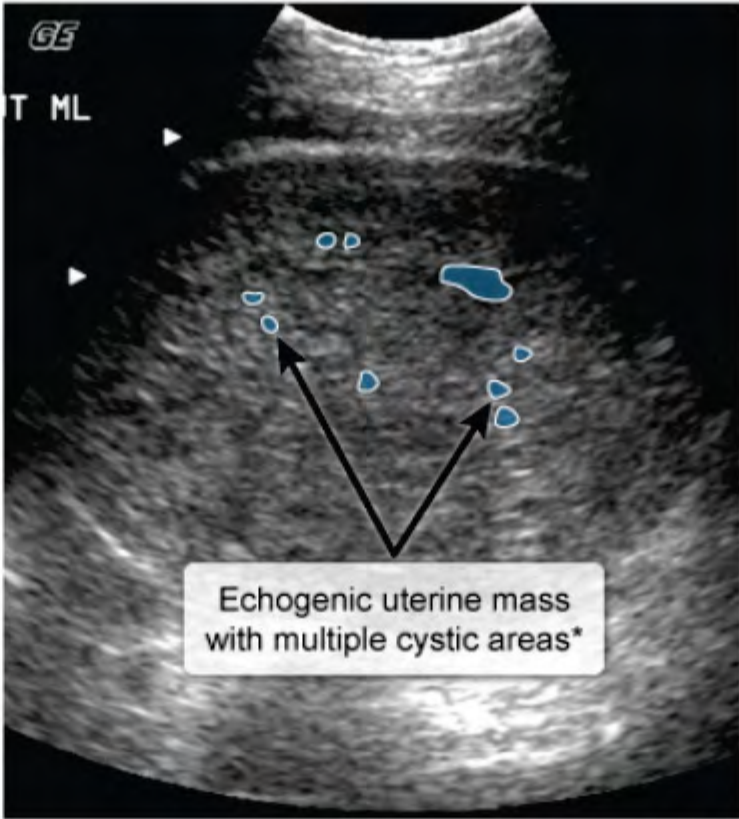
Exhibit Display

Complete hydatidiform mole



GE
T ML

***Snowstorm** appearance



GE
T ML

Echogenic uterine mass with multiple cystic areas*

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(Choice D) A miscarriage is a pregnancy loss prior to 20 weeks gestation and presents with vaginal bleeding and low β -hCG level. Pathology would show fetal tissue and normal villi and trophoblastic tissue.

(Choice F) A placental site trophoblastic tumor is a proliferation of intermediate trophoblasts. This malignant type

A 38-year-old woman, gravida 4 para 3, at 18 weeks gestation comes to the office for a routine anatomy ultrasound. The patient has had no vaginal bleeding, leakage of fluid, or contractions. She has no chronic medical conditions and her 3 prior pregnancies resulted in uncomplicated cesarean deliveries. Blood pressure is 118/66 mm Hg and pulse is 94/min. Fundal height is appropriate for gestational age. Transabdominal ultrasound reveals a fetus measuring 18 weeks gestation, an amniotic fluid index of 8 cm (normal: >5 cm), and an anterior placenta that covers the internal cervical os and penetrates the uterine myometrium. This patient is at greatest risk of placental invasion into which of the following structures?

- ☐ A. Bladder

☐ B. Broad ligament

☐ C. Cervix

☐ D. Infundibulopelvic ligament

☐ E. Uterosacral ligament

☐ F. Ureter

Submit

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- ✓

☐

A. Bladder (43%)
- ✗

☒

B. Broad ligament (8%)
- ☐

C. Cervix (35%)
- ☐

D. Infundibulopelvic ligament (4%)
- ☐

E. Uterosacral ligament (5%)
- ☐

F. Ureter (2%)

Incorrect

Correct answer
A

43%
Answered correctly

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Explanation



A 38-year-old woman, gravida 4, para 2, at 18 weeks gestation comes to the office for a routine anatomy ultrasound.

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Placenta percreta bladder invasion

Placenta

Prior uterine scar

Bladder

Chorionic villi invasion

Hematuria

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This patient has **placental accreta spectrum**, a disorder defined by the depth of abnormal placental invasion: placenta accreta (attachment to the myometrium), placenta increta (invasion into the myometrium), and placenta percreta (invasion through the myometrium and uterine serosa and into extrauterine structures).

The most common site for abnormal placental implantation is over a prior **uterine scar**; these areas have thin myometrium and lack the decidua basalis layer needed to form a normal uteroplacental interface. Patients with **repeat cesarean deliveries** commonly have an anterior, low transverse scar; therefore, placental implantation in future pregnancies is typically anterior and low, which can result in the placenta covering the internal cervical os (ie, **placenta previa**). Eventually, an invasive anterior placenta can separate the uterine scar and invade across the uterine wall into adjacent structures (ie, **placenta percreta**). The structure immediately anterior to the uterus is the bladder, so patients with an **anterior** placenta percreta are at greatest risk for **bladder invasion**.

(Choice B) The **broad ligament** is a wide fold of peritoneum containing the round ligament, the uteroovarian ligament, and the fallopian tube. Because it extends laterally from the uterus to the pelvic sidewall, it is at lower risk for invasion by an anterior placenta.

(Choice C) Although placentas can implant over the internal cervical os, placental invasion of the cervix does not usually occur because the cervix is composed of dense connective tissue and is much firmer, less vascular, and less vulnerable to penetration compared to the myometrium.

(Choice D) The **infundibulopelvic ligament** (ie, suspensory ligament of the ovary), which contains the ovarian vessels, connects the ovary to the pelvic sidewall. Because it is lateral to the uterus, it is unlikely to be affected by placental invasion.

(Choice E) The **uterosacral ligament** anchors the posterior uterus to the sacrum for pelvic support. Because it attaches to the posterior uterus, it is unlikely to be invaded by an anterior placenta.

(Choice F) The ureter travels along the lateral pelvic sidewall before diving posterior to the uterine vessels and entering the bladder. Its path is posterior and lateral to the uterus, making placental invasion by an anterior

myometrium and lack the decidual basalis layer needed to form a normal uteroplacental interface. Patients with **repeat cesarean deliveries** commonly have an anterior, low transverse scar; therefore, placental implantation in future pregnancies is typically anterior and low, which can result in the placenta covering the internal cervical os (ie, **placenta previa**). Eventually, an invasive anterior placenta can separate the uterine scar and invade across the uterine wall into adjacent structures (ie, **placenta percreta**). The structure immediately anterior to the uterus is the bladder, so patients with an **anterior** placenta percreta are at greatest risk for **bladder invasion**.

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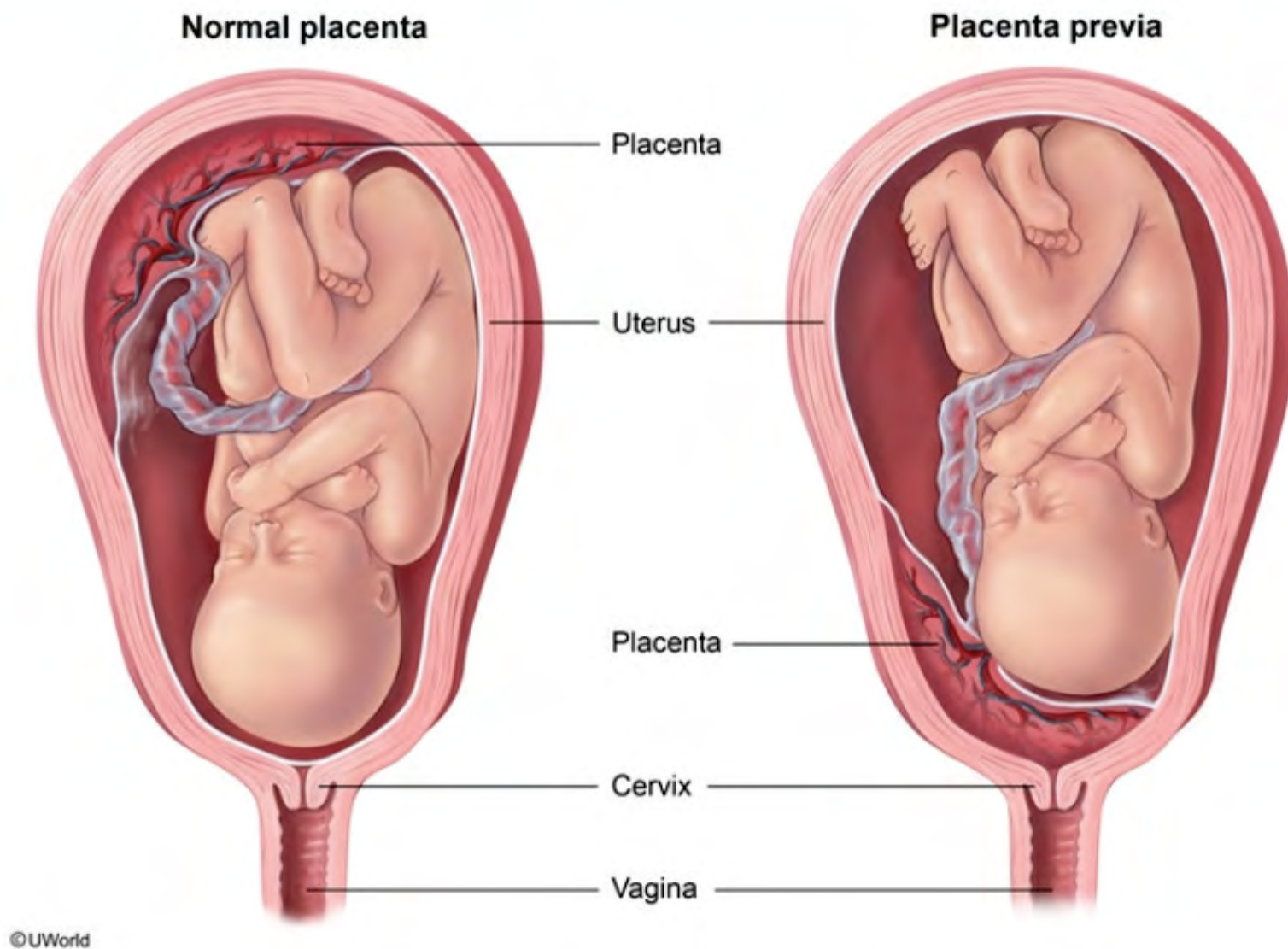
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(Choice F) The ureter travels along the lateral pelvic sidewall before diving posterior to the uterine vessels and entering the bladder. Its path is posterior and lateral to the uterus, making placental invasion by an anterior placenta unlikely.

Educational objective:
Placenta percreta is placental invasion through the myometrium and uterine serosa and into extrauterine structures. An anterior placenta percreta is most likely to invade structures anterior to the uterus such as the bladder.

Prior uterine

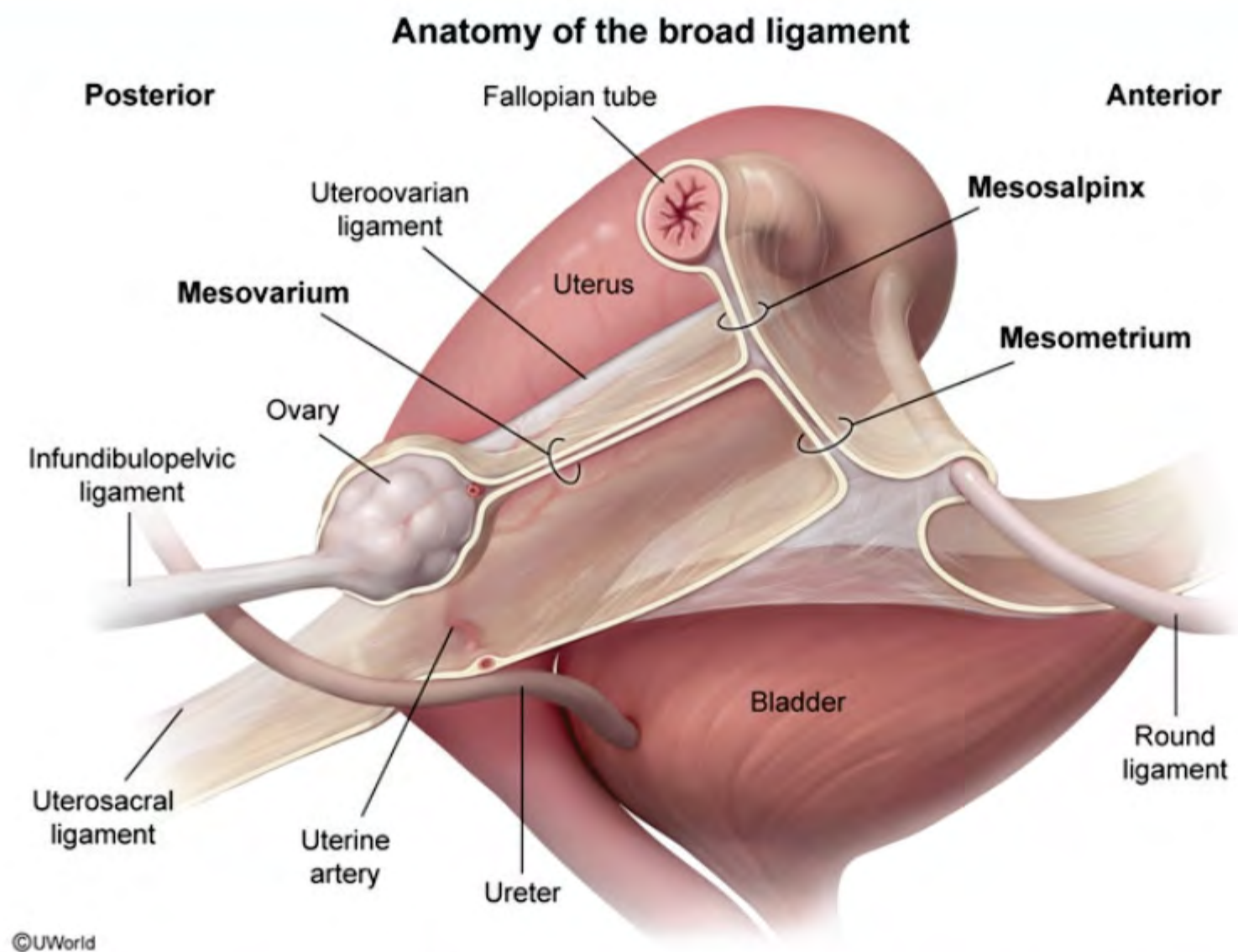
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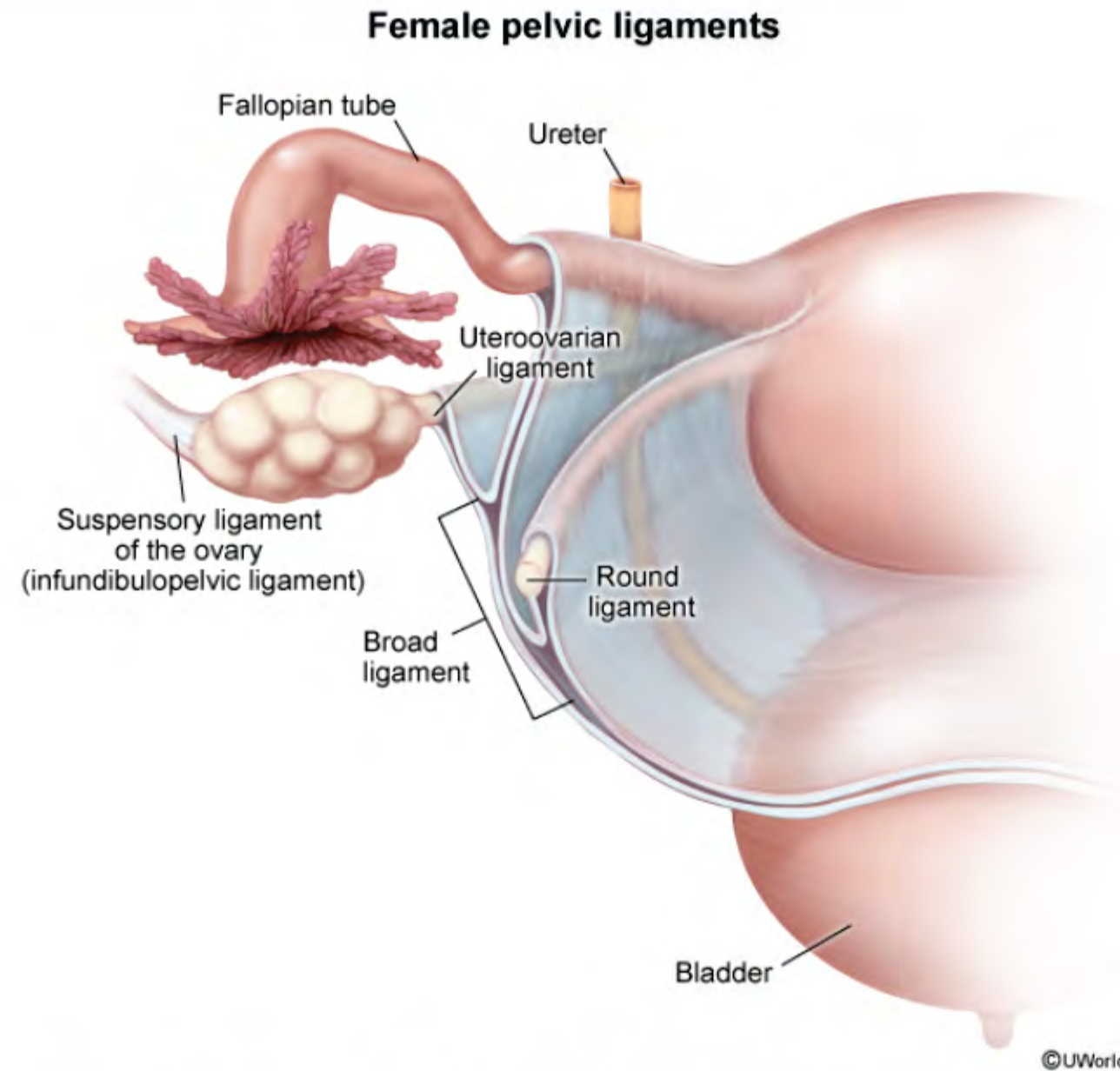
This placental invasion. The myometrium is repeated in the future (ie, uterine bladder). (Choice A) ligament risk. (Choice B) usual less. (Choice C)

vessels, connects the ovary to the pelvic sidewall. Because it is lateral to the uterus, it is unlikely to be affected by placental invasion.

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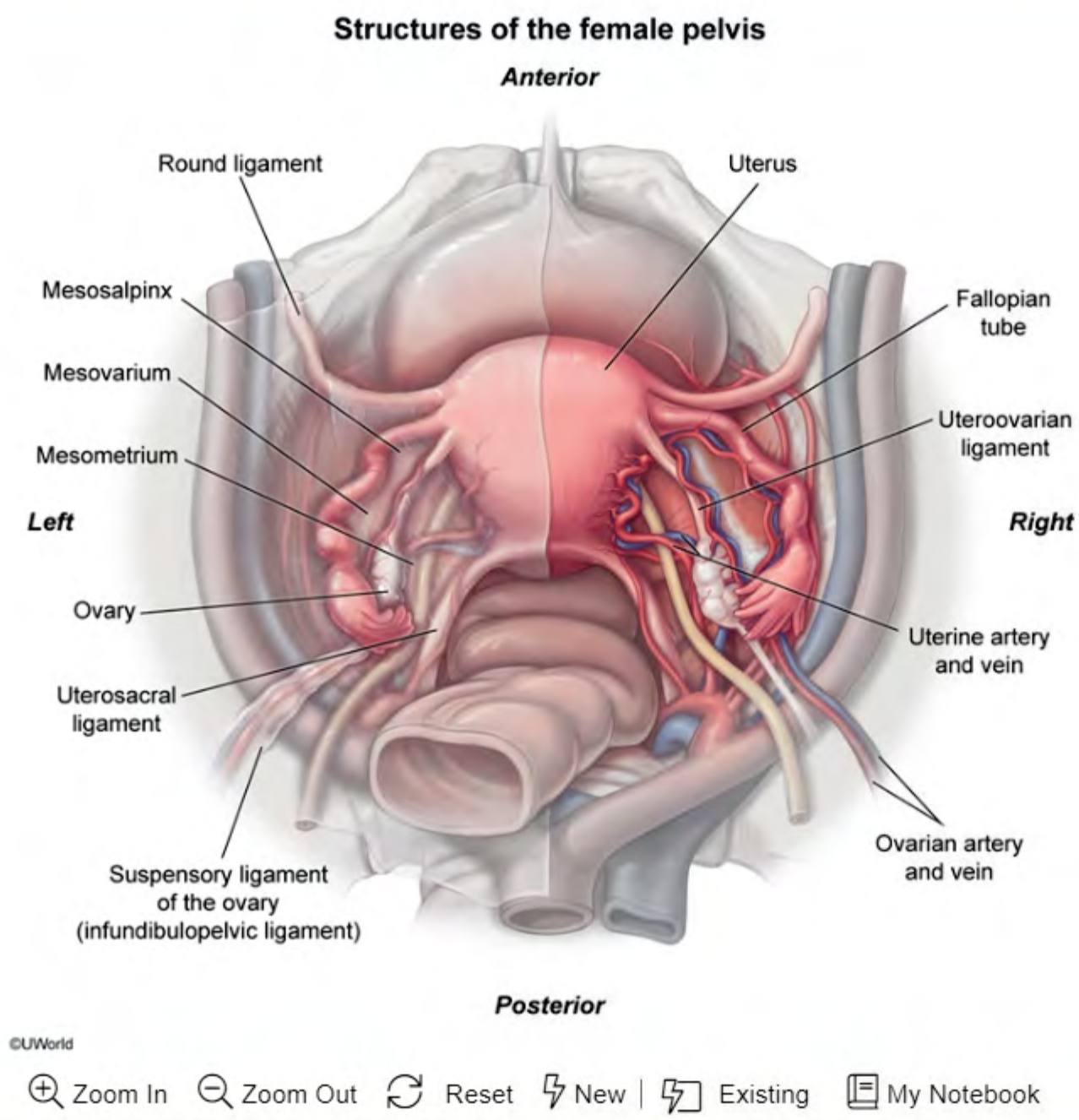


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percreta (invasion through the myometrium and uterine serosa and into extrauterine structures).

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Educational objective:

A 24-year-old primigravid woman at 36 weeks gestation comes to the office due to light-headedness and nausea at bedtime. Her pregnancy has been uncomplicated, and medical history is unremarkable. The patient takes folic acid daily but has been unable to tolerate oral iron. Blood pressure is 115/75 mm Hg when sitting, 110/70 mm Hg when standing, and 80/60 mm Hg when supine. Physical examination shows a uterus consistent in size with 36 weeks gestation. Which of the following is the most likely explanation for this patient's hypotension while supine?

- ☐ A. Decreased venous return
- ☐ B. Increased hemodilution
- ☐ C. Increased parasympathetic activity
- ☐ D. Reduced peripheral arterial resistance
- ☐ E. Volume depletion

Submit

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- ✓

☐

A. Decreased venous return (69%)
- ✗

☒

B. Increased hemodilution (5%)
- ☐

C. Increased parasympathetic activity (6%)
- ☐

D. Reduced peripheral arterial resistance (16%)
- ☐

E. Volume depletion (1%)

Incorrect

Correct answer
A

69%

Answered correctly

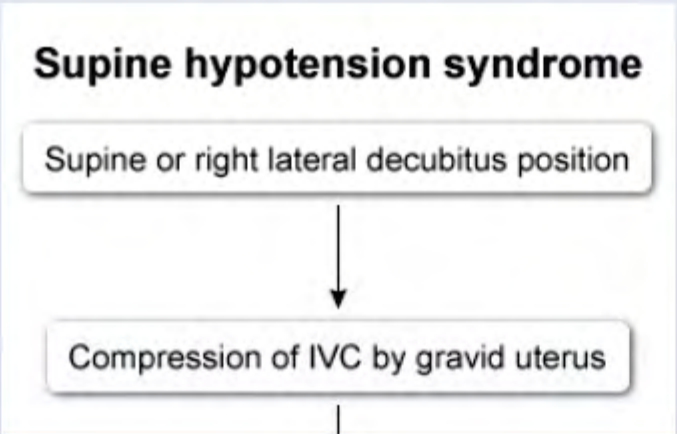
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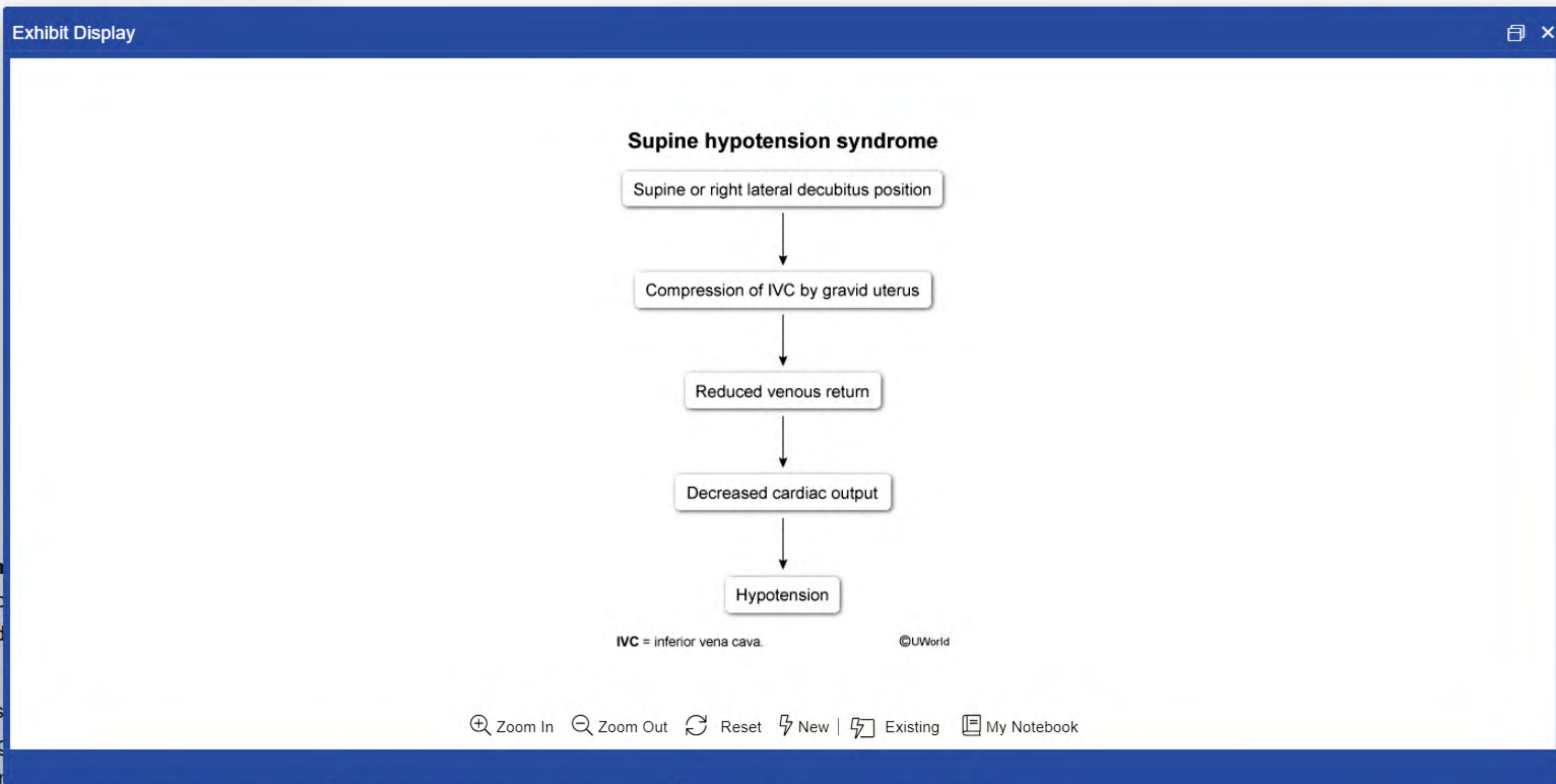
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Explanation





Compression of the **inferior vena cava** (IVC) occurs in most pregnancies and begins when the gravid uterus reaches the level of the umbilicus (ie, 20 weeks gestation). The degree of obstruction is most prominent during the third trimester and is worse when the mother lies in the **supine** or **right lateral decubitus** position.

IVC compression **reduces venous return** (ie, cardiac preload), which subsequently **decreases cardiac output**. Most patients compensate for this physiologic change with increased collateral blood flow primarily through the azygous vein (connecting the iliac veins to the superior vena cava), which completely bypasses the IVC and maintains venous return. Because of significant anatomic variation of the azygous vein, some lack this robust collateral circulation. These patients are particularly sensitive to the decrease in venous return occurring in the supine or right lateral decubitus position, a condition known as **supine hypotension syndrome** (or aortocaval compression syndrome).

A significant drop in blood pressure can develop and/or heart rate can increase when patients with supine hypotension syndrome are placed in the supine position; corresponding symptoms include pallor, sweating, nausea, and light-headedness. In severe cases, loss of consciousness and even fetal demise can occur.

(Choices B and E) Hemodilution is normal in pregnancy because of a relatively greater physiologic increase in plasma volume compared to red blood cell mass. Hemodilution may be increased in patients who cannot tolerate oral iron but is unlikely to significantly contribute to hypotension. True intravascular volume depletion is a common cause of hypotension, which worsens with standing and tends to improve with supine positioning (ie, orthostatic hypotension).

(Choice C) Increased parasympathetic activity drives the hypotension that occurs with a vasovagal response. Such a response is typically triggered by emotional stress or prolonged standing but not by supine positioning.

(Choice D) Peripheral arterial resistance is reduced beginning in early pregnancy due to rising progesterone levels. This causes a slight decrease in blood pressure but does not explain isolated supine hypotension.

Educational objective:

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Educational objective:

Patients at >20 weeks gestation experience compression of the inferior vena cava by the gravid uterus while in the supine or right lateral decubitus position. Without substantial collateral blood flow, the consequent reduction in venous return and cardiac output can cause severe hypotension (supine hypotension syndrome).

A 34-year-old woman, gravida 0, comes to the office for evaluation of pregnancy. Menarche was at age 12, and her menstrual cycles are regular; they occur every 30 days with 5 days of menses. The patient and her husband have been trying to conceive for the past 6 months and plan intercourse during her fertile window according to an ovulation predictor kit. She has no medical conditions, takes no medications, and has no allergies. BMI is 23 kg/m². Vital signs and physical examination are normal. If fertilization and implantation occurred in this cycle, when would the β -hCG level first be detectable in the serum?

- ☐ A. 1 day after fertilization
- ☐ B. 3 days after fertilization
- ☐ C. 8 days after fertilization
- ☐ D. 14 days after fertilization
- ☐ E. On the day of fertilization

Submit

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- A. 1 day after fertilization (2%)

✖

B. 3 days after fertilization (12%)

✔

C. 8 days after fertilization (66%)

D. 14 days after fertilization (18%)

E. On the day of fertilization (1%)

Incorrect

Correct answer C

66%

Answered correctly

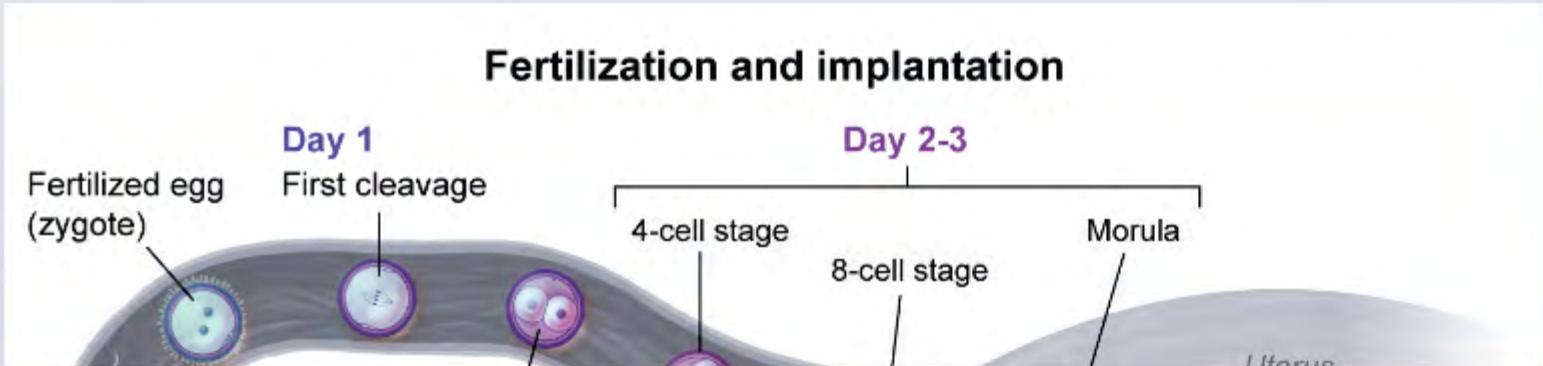
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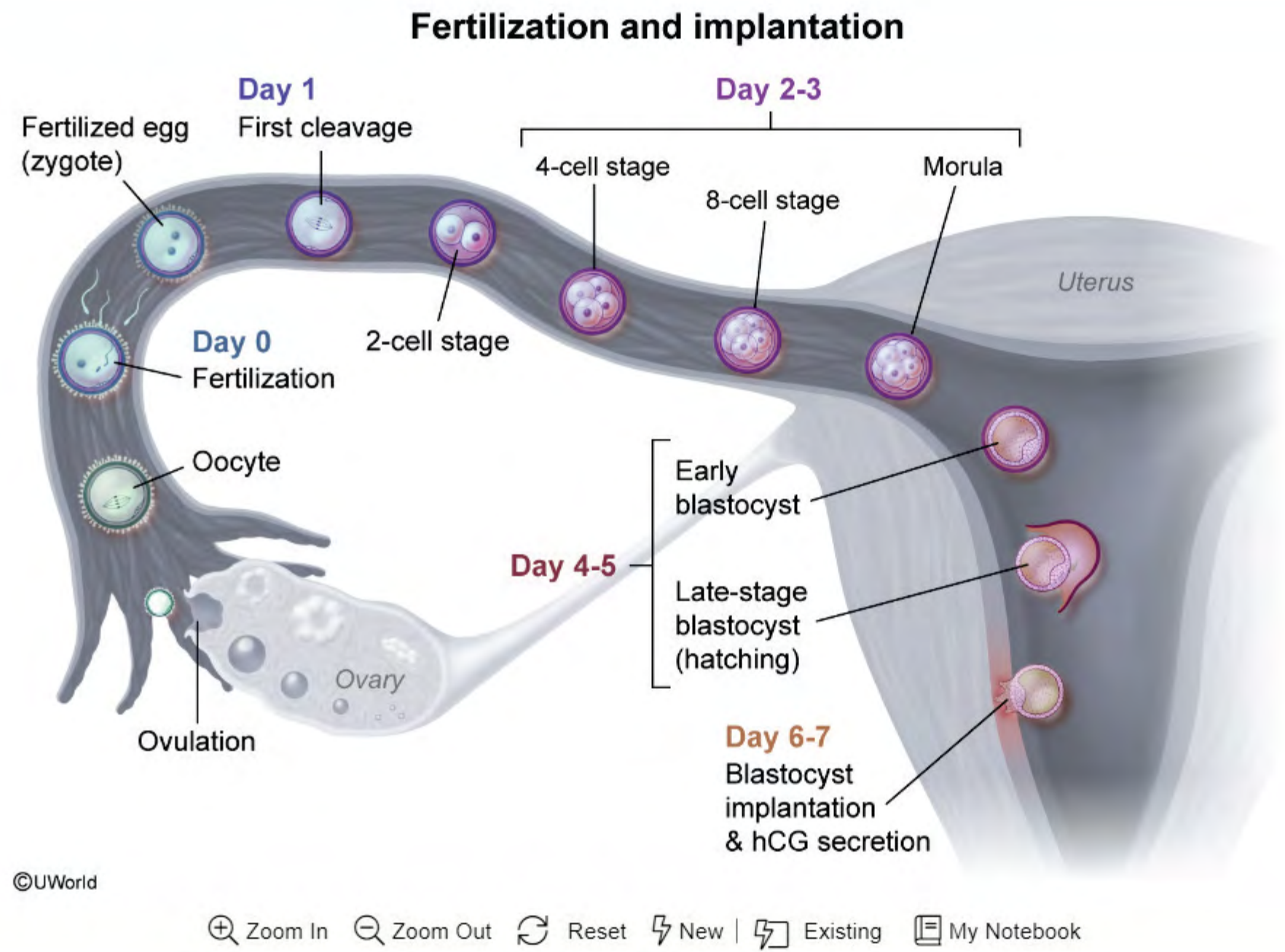
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Explanation



Explanation

Exhibit Display



The morula enters the uterus 3-4 days after fertilization and forms a central cavity, converting it into a blastocyst.

Timing intercourse around a woman's fertility window can help couples achieve pregnancy. Ovulation predictor kits measure urinary luteinizing hormone levels, and the tests are positive 24 hours before ovulation.

Once an oocyte is released from the ovary, sperm may fertilize it for up to 24 hours. Following fertilization, the second meiotic division completes, forming a zygote that travels through the fallopian tube while undergoing multiple mitotic divisions (ie, cleavage) and creating smaller cells (blastomeres) known collectively as a morula. The morula enters the uterus 3-4 days after fertilization and forms a central cavity, converting it into a blastocyst.

The blastocyst **implants** about 6 days after fertilization (**Choices A, B, and E**), and the outer cell mass (trophoblast) differentiates into the cytotrophoblast and **syncytiotrophoblast**. The syncytiotrophoblast invades the endometrial connective tissue 6-7 days after fertilization and begins **secreting β -hCG**, which enters the maternal circulation and maintains corpus luteum progesterone secretion.

Accordingly, β -hCG may appear in the maternal serum as early as 6 days after fertilization; however, additional time (typically **8 days**) is often required for the serum to rise to a detectable level. β -hCG is detectable in the maternal serum at <5 IU/L, making **serum β -hCG** testing the **most sensitive** method of detecting pregnancy.







(Choice D) A urine pregnancy test is less sensitive than a serum pregnancy test; it is positive when β -hCG reaches 20 IU/L, which typically occurs 14 days following fertilization. Therefore, a serum pregnancy test will be positive before a urine pregnancy test.

Educational objective:

β -hCG is produced by the syncytiotrophoblast after implantation, which generally occurs 6-7 days after fertilization at the earliest. β -hCG typically is detectable in the maternal serum 8 days after fertilization, whereas it is detectable in the urine 14 days after fertilization.



*Hyperglycosylated hCG

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A 36-year-old woman, gravida 2 para 1, at 38 weeks gestation comes to the hospital in active labor, dilated to 10 cm. She has received no prenatal care this pregnancy. Her prior pregnancy resulted in an uncomplicated cesarean delivery. On admission, temperature is 36.7 C (98.1 F), blood pressure is 132/84 mm Hg, and pulse is 94/min. The patient precipitously delivers an infant weighing 4.1 kg (9 lb 2 oz). After delivery of the infant, small placental fragments are removed in pieces via manual extraction. Profuse vaginal bleeding occurs, and intravenous lines are placed. Uterotonic medications are administered and vigorous uterine massage is performed. The uterine fundus is firm, but the bleeding continues. Which of the following is the most likely cause of this patient's ongoing vaginal bleeding?

- ☐ A. Full-thickness rupture of the uterine myometrium
- ☐ B. Hypertension-induced placental detachment
- ☐ C. Inadequate uterine contraction
- ☐ D. Infection of the uterus and placental membranes
- ☐ E. Placental invasion into the uterine myometrium

Submit

A 36-year-old woman, gravida 2 para 1, at 38 weeks gestation comes to the hospital in active labor, dilated to 10 cm. She has received no prenatal care this pregnancy. Her prior pregnancy resulted in an uncomplicated cesarean delivery. On admission, temperature is 36.7 C (98.1 F), blood pressure is 132/84 mm Hg, and pulse is 94/min. The patient precipitously delivers an infant weighing 4.1 kg (9 lb 2 oz). After delivery of the infant, small placental fragments are removed in pieces via manual extraction. Profuse vaginal bleeding occurs, and intravenous lines are placed. Uterotonic medications are administered and vigorous uterine massage is performed. The uterine fundus is firm, but the bleeding continues. Which of the following is the most likely cause of this patient's ongoing vaginal bleeding?

- A. Full-thickness rupture of the uterine myometrium (10%)

✗

B. Hypertension-induced placental detachment (2%)

C. Inadequate uterine contraction (11%)

D. Infection of the uterus and placental membranes (1%)

✓

E. Placental invasion into the uterine myometrium (74%)
- Incorrect

Correct answer
E

74%

Answered correctly

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Version

Explanation

The diagram shows a cross-section of the uterus and placenta. The placenta is shown attached to the uterine wall, with the label "Placenta accreta" above it. The placenta is shown as a mass of red and pink tissue, with the label "Placenta accreta" above it.

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My Notebook

Flashcards

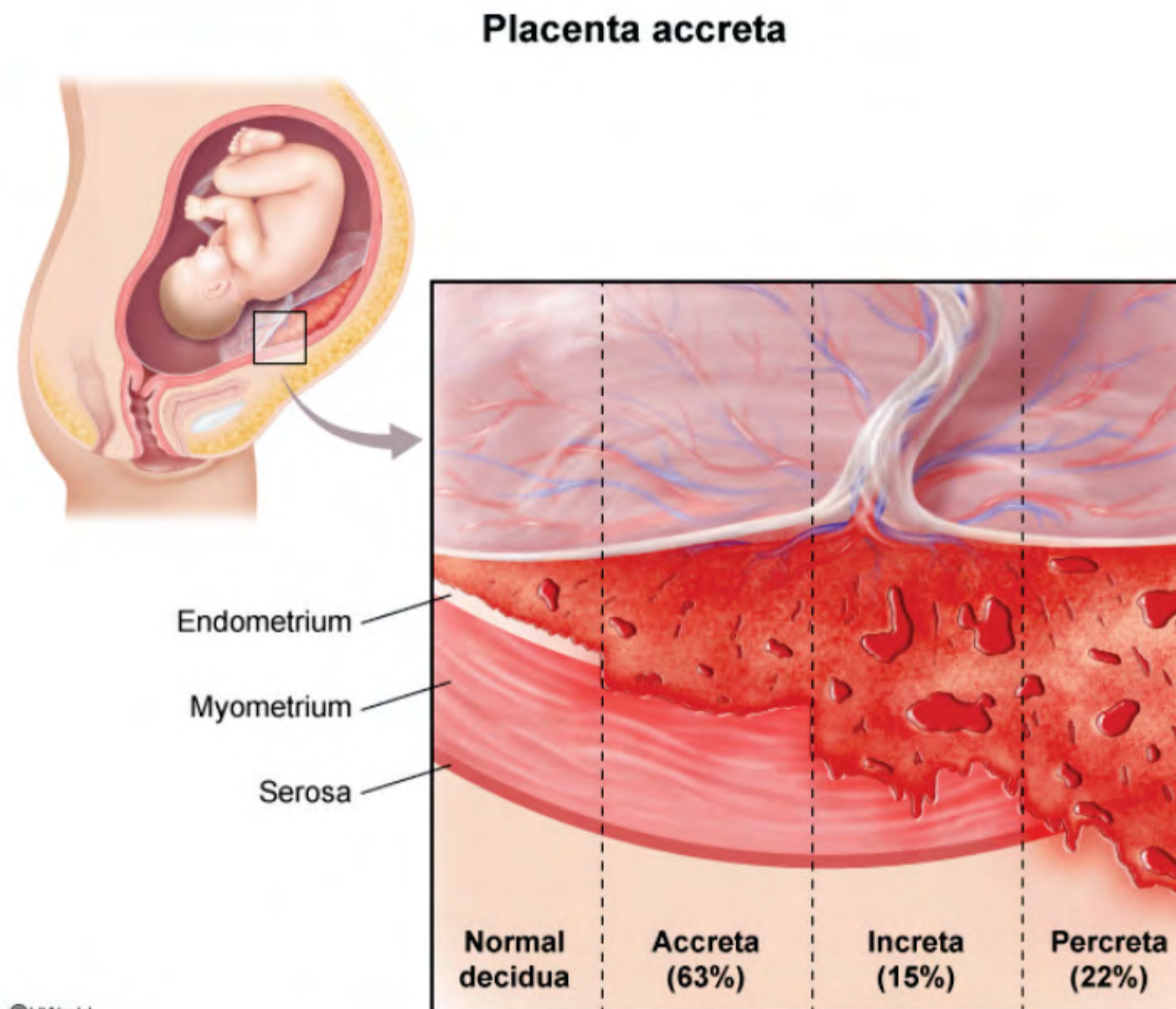
Feedback

Suspend

End Block

☐ A. Full-thickness rupture of the uterine myometrium (10%)

Exhibit Display



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Zoom In Zoom Out Reset New Existing My Notebook

This patient's **postpartum hemorrhage** (PPH) is likely due to **placental invasion into the myometrium**, which characterizes **placenta accreta** spectrum. The disease range is defined by depth of placental invasion: placenta accreta (attachment to the myometrium), placenta increta (invasion into the myometrium), and placenta percreta (invasion through the myometrium and serosa).

Placenta accreta disorders occur due to placental invasion through **defects in the decidua basalis** of the endometrium, which are commonly caused by uterine scarring from prior uterine surgery (eg, **cesarean delivery**, dilation and curettage). The absence of an intact decidual layer and subsequent direct placental attachment to the myometrium makes for a difficult placental delivery. Usually, manual placental extraction is attempted but yields only **small placental fragments** due to dense adhesions between the placenta and the uterus that **bleed profusely** if injured.

(Choice A) **Uterine rupture**, a full-thickness myometrial disruption, can cause vaginal bleeding; however, patients typically have antepartum (not postpartum) bleeding and intense abdominal pain. It would not cause difficulty with placental detachment.

(Choice B) **Abruptio placentae** is premature placental separation from the uterus; it can occur secondary to hypertension-induced placental vessel rupture. Unlike placenta accreta, abruptio placentae causes antepartum bleeding and promotes, rather than delays, placental detachment.

(Choice C) Uterine atony (ie, poor uterine contractility) is the most common cause of PPH and typically presents with a soft, boggy uterine fundus. This patient's firm uterine fundus and unresponsiveness to uterotonic medications (which contract the uterus) make this diagnosis less likely.

(Choice D) Intraamniotic infection can cause vaginal bleeding and an inflamed, adherent placenta. However, the anatomic plane between the placenta and uterus remains intact and therefore does not cause placental fragmentation. In addition, this patient has no fever or purulent amniotic fluid, making this diagnosis unlikely.

Educational objective:

(invasion through the myometrium and serosa).

Placenta accreta disorders occur due to placental invasion through **defects in the decidua basalis** of the endometrium, which are commonly caused by uterine scarring from prior uterine surgery (eg, **cesarean delivery**, dilation and curettage). The absence of an intact decidual layer and subsequent direct placental attachment to the myometrium makes for a difficult placental delivery. Usually, manual placental extraction is attempted but yields only **small placental fragments** due to dense adhesions between the placenta and the uterus that **bleed profusely** if injured.

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Educational objective:

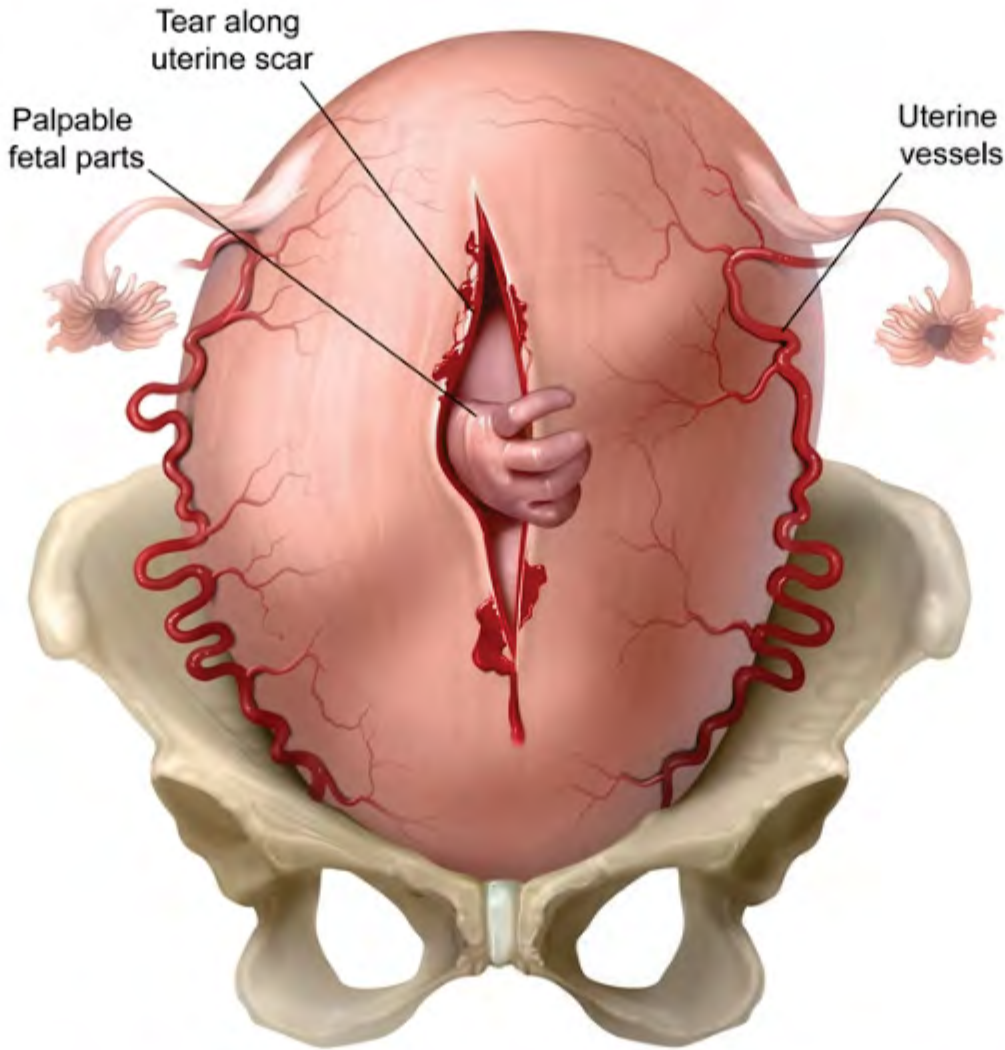
Placenta accreta spectrum occurs due to placental invasion into the myometrium through defects in the decidua basalis. This creates a morbidly adherent placenta that does not detach after fetal delivery, leading to postpartum hemorrhage. Manual placental extraction typically yields small placental fragments and increases bleeding.

References

This patient's **postpartum hemorrhage** (PPH) is likely due to **placental invasion into the myometrium**, which

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Uterine rupture



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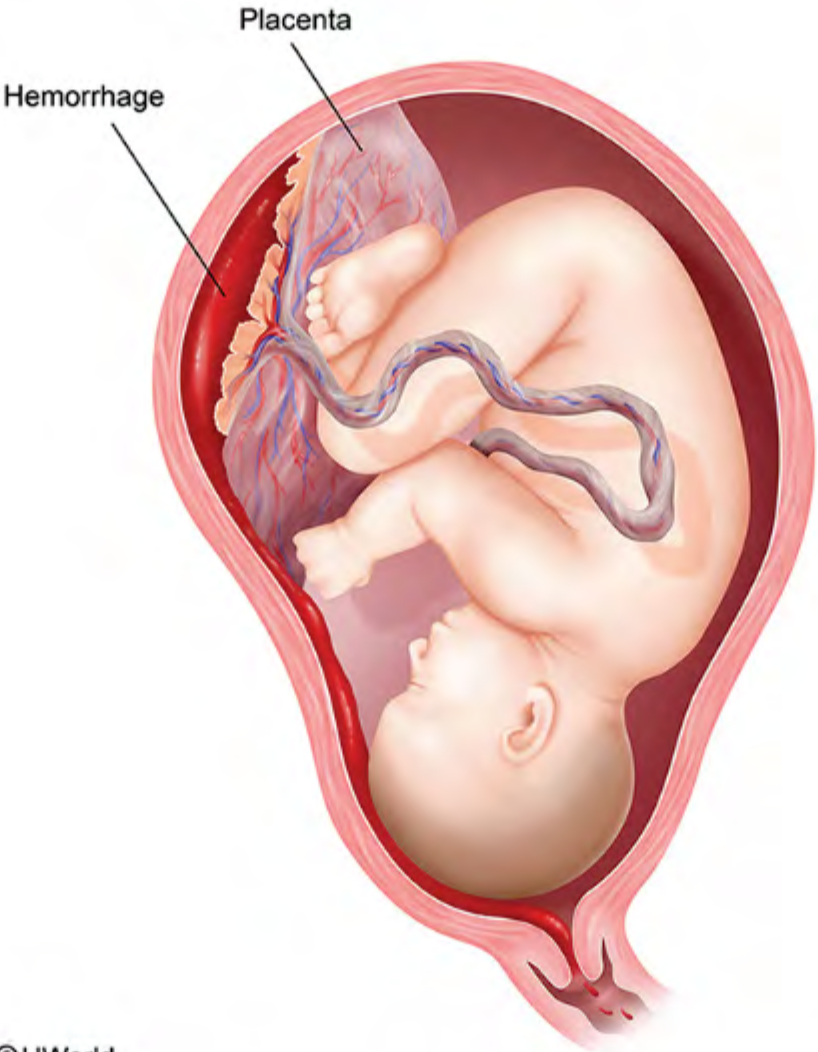
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Placenta accreta spectrum occurs due to placental invasion into the myometrium through defects in the decidua

This patient's **postpartum hemorrhage** (PPH) is likely due to **placental invasion into the myometrium**, which

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Placental abruption



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Placenta accreta spectrum occurs due to placental invasion into the myometrium through defects in the decidua

A 7-year-old boy is evaluated for fatigue. For the last year, he has been avoiding any significant outdoor activities with his peers due to poor energy and shortness of breath. He also describes occasional pounding in the chest. Physical examination shows a holosystolic murmur best heard at the lower sternal border. Echocardiography reveals apical displacement of the tricuspid valve leaflets, decreased right ventricular volume, and atrialization of the right ventricle. Moderate to severe tricuspid regurgitation is also present. If this patient's diagnosis is due to a side effect of a drug taken during pregnancy, his biological mother most likely had which of the following conditions?

- ☐ A. Alcohol use disorder
- ☐ B. Bipolar disorder
- ☐ C. Cocaine use disorder
- ☐ D. Down syndrome
- ☐ E. Epilepsy
- ☐ F. Gestational diabetes
- ☐ G. Hypothyroidism
- ☐ H. Opioid use disorder
- ☐ I. Schizophrenia

Submit

A 7-year-old boy is evaluated for fatigue. For the last year, he has been avoiding any significant outdoor activities with his peers due to poor energy and shortness of breath. He also describes occasional pounding in the chest. Physical examination shows a holosystolic murmur best heard at the lower sternal border. Echocardiography reveals apical displacement of the tricuspid valve leaflets, decreased right ventricular volume, and atrialization of the right ventricle. Moderate to severe tricuspid regurgitation is also present. If this patient's diagnosis is due to a side effect of a drug taken during pregnancy, his biological mother most likely had which of the following conditions?

- A. Alcohol use disorder (4%)

✓

☒ B. Bipolar disorder (74%)

C. Cocaine use disorder (3%)

D. Down syndrome (0%)

E. Epilepsy (9%)

F. Gestational diabetes (2%)

G. Hypothyroidism (1%)

H. Opioid use disorder (0%)

I. Schizophrenia (2%)

Correct

74%
Answered correctly

03 secs
Time Spent

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Explanation

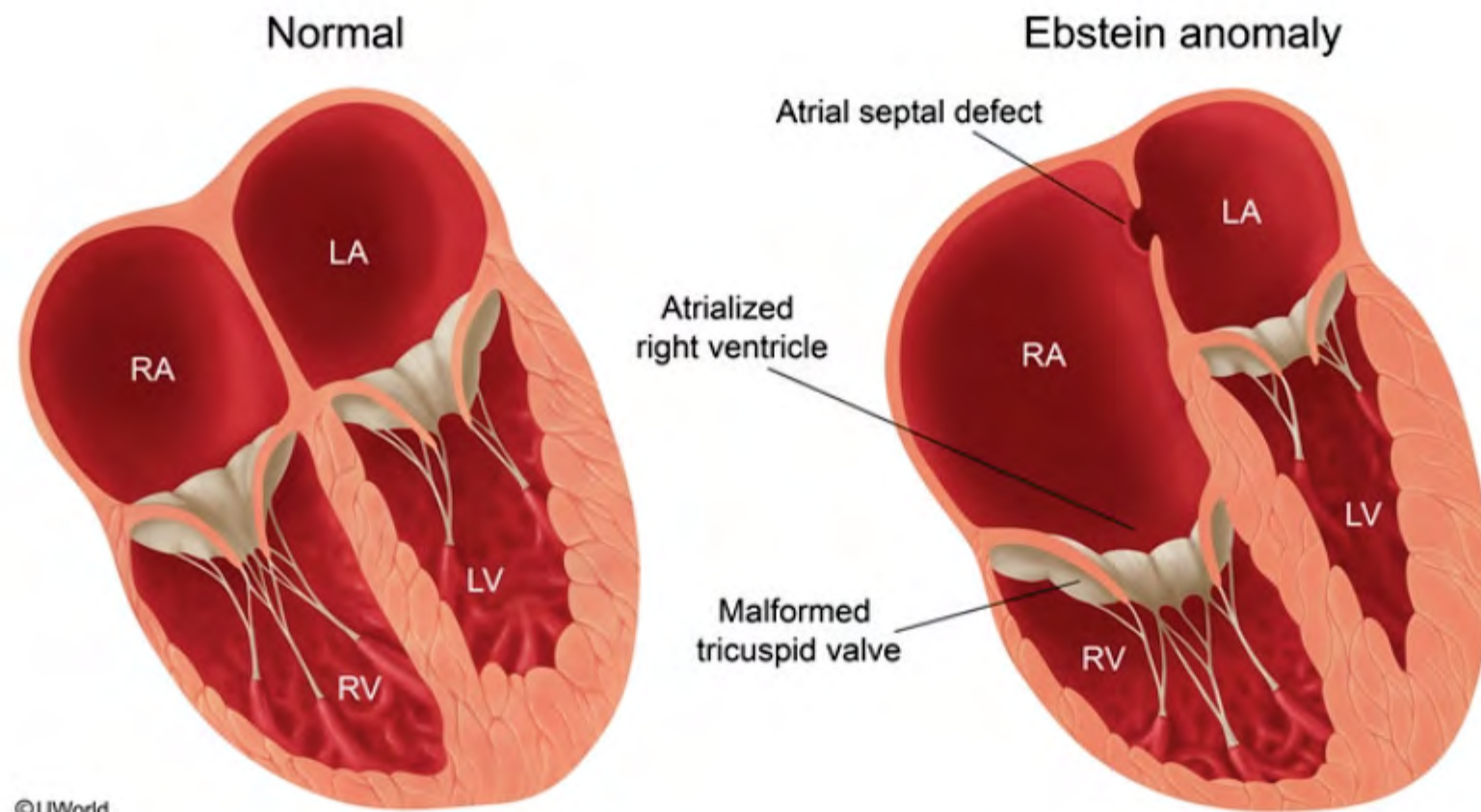
Correct

74%

03 secs

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Exhibit Display



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dysmorphic facies, growth retardation, and central nervous system abnormalities.

In patients with **bipolar disorder**, **lithium** is used to treat acute mania, hypomania, and bipolar and unipolar depression. Long-term treatment may also reduce the risk of suicide attempts and deaths. Lithium is potentially teratogenic; in utero exposure is associated with Ebstein's anomaly in infants. Ebstein's anomaly is characterized by apical displacement of the tricuspid valve leaflets, decreased volume of the right ventricle, and atrialization of the right ventricle.

(Choice A) Alcohol use during pregnancy may result in **fetal alcohol syndrome**, which is characterized by dysmorphic facies, growth retardation, and central nervous system abnormalities.

(Choice C) Prenatal cocaine use increases the risk of preeclampsia, spontaneous abortion, fetal demise, and placental abruption.

(Choice D) Atrial and ventricular septal defects are the most common congenital cardiac anomalies in patients with **Down syndrome**.

(Choice E) Seizures are not associated with major fetal malformations, but antiepileptic drugs (AEDs) significantly increase the risk of **neural tube**, renal, skeletal, and cleft palate abnormalities. Although AEDs also increase the risk of congenital heart defects, they are not associated with Ebstein's anomaly.

(Choice F) Gestational diabetes is associated with fetal macrosomia, caudal regression syndrome, hypoglycemia, hypocalcemia, and hypertrophic cardiomyopathy.

(Choice G) Untreated maternal hypothyroidism in pregnancy is associated with obstetric complications and cognitive impairment in offspring.

(Choice H) Opioid use may cause neonatal withdrawal symptoms (irritability, diarrhea, vomiting) after delivery. Opioid dependence increases the risk of multiple obstetric complications but not major cardiac defects.

(Choice I) Schizophrenia and antipsychotics are not associated with the development of major fetal malformations.

Educational objective:

the right ventricle.

- (Choice A)** Alcohol use during pregnancy may result in fetal alcohol syndrome, which is characterized by dysmorphic facies, growth retardation, and central nervous system abnormalities.
- (Choice C)** Prenatal cocaine use increases the risk of preeclampsia, spontaneous abortion, fetal demise, and placental abruption.
- (Choice D)** Atrial and ventricular septal defects are the most common congenital cardiac anomalies in patients with Down syndrome.
- (Choice E)** Seizures are not associated with major fetal malformations, but antiepileptic drugs (AEDs) significantly increase the risk of neural tube, renal, skeletal, and cleft palate abnormalities. Although AEDs also increase the risk of congenital heart defects, they are not associated with Ebstein's anomaly.
- (Choice F)** Gestational diabetes is associated with fetal macrosomia, caudal regression syndrome, hypoglycemia, hypocalcemia, and hypertrophic cardiomyopathy.
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- (Choice H)** Opioid use may cause neonatal withdrawal symptoms (irritability, diarrhea, vomiting) after delivery. Opioid dependence increases the risk of multiple obstetric complications but not major cardiac defects.
- (Choice I)** Schizophrenia and antipsychotics are not associated with the development of major fetal malformations.

Educational objective:

Lithium is commonly used to treat bipolar disorder. Its use during pregnancy is associated with Ebstein's anomaly, which is characterized by apical displacement of the tricuspid valve leaflets, decreased right ventricular volume, and atrialization of the right ventricle.

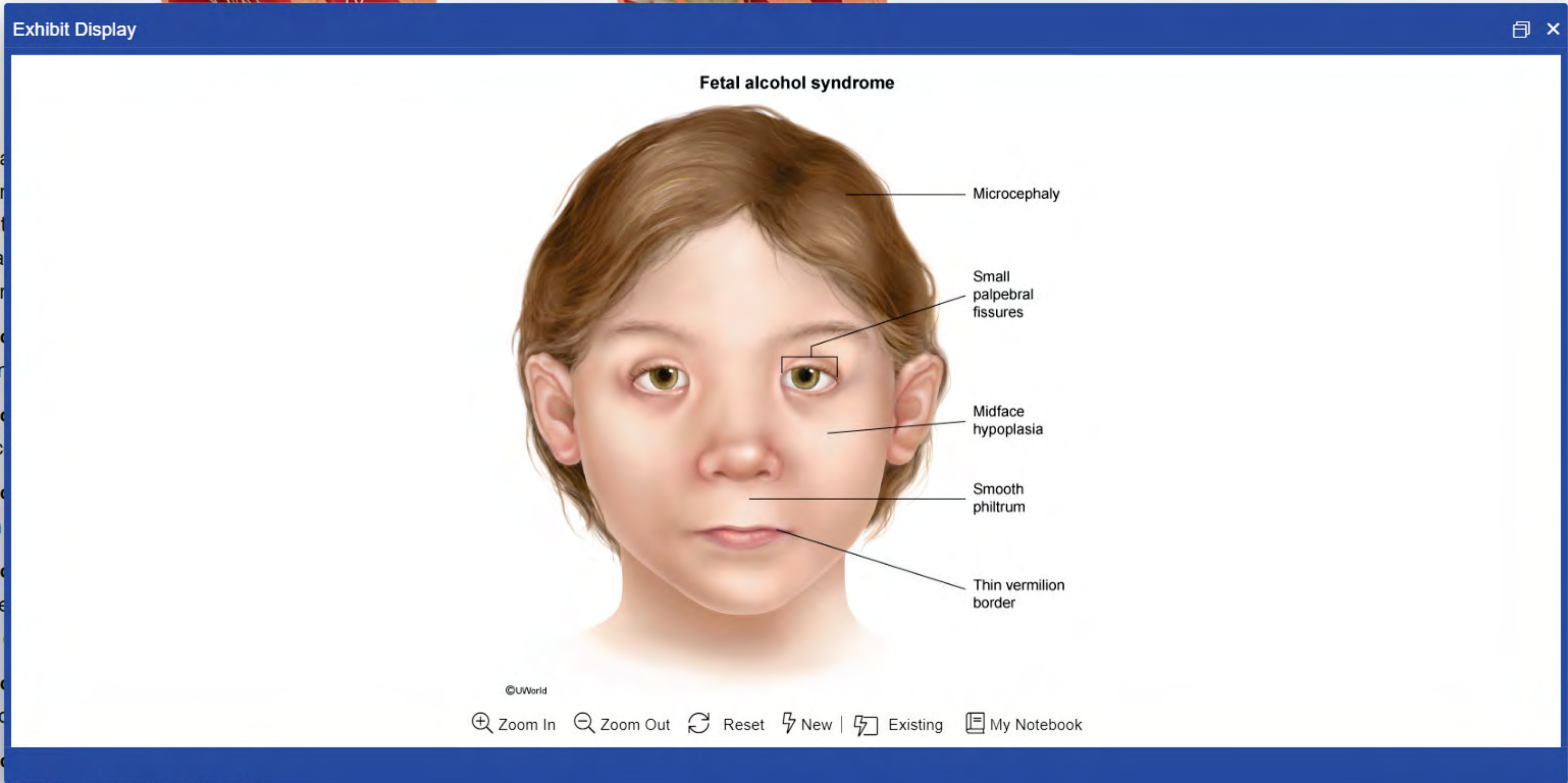


Exhibit Display

Down syndrome comorbidities	
Neurologic	<ul style="list-style-type: none">Intellectual disabilityEarly-onset Alzheimer disease
Cardiac	<ul style="list-style-type: none">Complete atrioventricular septal defectVentricular septal defectAtrial septal defect
Gastrointestinal	<ul style="list-style-type: none">Duodenal atresiaHirschsprung disease
Endocrine	<ul style="list-style-type: none">HypothyroidismType 1 diabetes mellitusObesityShort stature
Oncologic	<ul style="list-style-type: none">Acute leukemia
Orthopedic	<ul style="list-style-type: none">Atlantoaxial instability

New

Existing

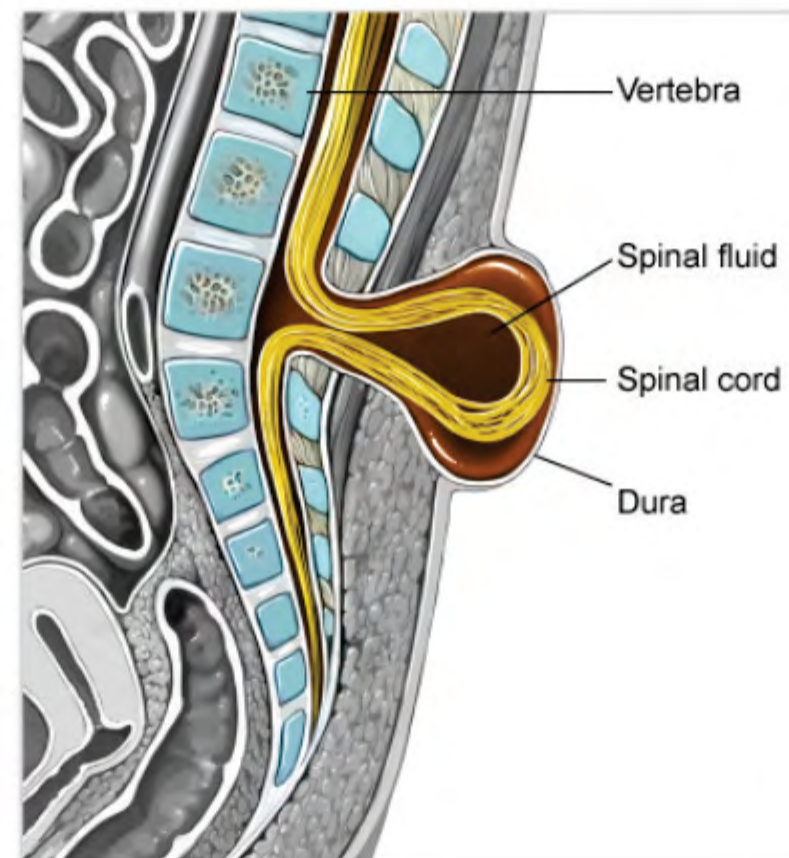
My Notebook

Exhibit Display

Open spina bifida



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A 10-minute-old boy is being evaluated in the delivery room. The patient was born at term to a 30-year-old primigravida woman. Pregnancy was unremarkable, but labor was complicated by recurrent late decelerations necessitating vacuum assistance for vaginal delivery. Apgar scores were 6 and 8 at 1 and 5 minutes, respectively. Temperature is 37 C (98.6 F), pulse is 170/min, and respirations are 40/min. Scalp examination shows large, fluctuant swelling at the occiput that extends bilaterally to the ears, superiorly toward the crown, and inferiorly into the nape of the neck. This patient's hemorrhage is most likely located between which of the following structures?

- ☐ A. Arachnoid mater and dura mater
- ☐ B. Brain and arachnoid mater
- ☐ C. Dura mater and periosteum
- ☐ D. Periosteum and galea aponeurosis
- ☐ E. Skull and the periosteum

Submit

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- A. Arachnoid mater and dura mater (23%)

✗

B. Brain and arachnoid mater (9%)

C. Dura mater and periosteum (19%)

✓

D. Periosteum and galea aponeurosis (30%)

E. Skull and the periosteum (16%)

Incorrect

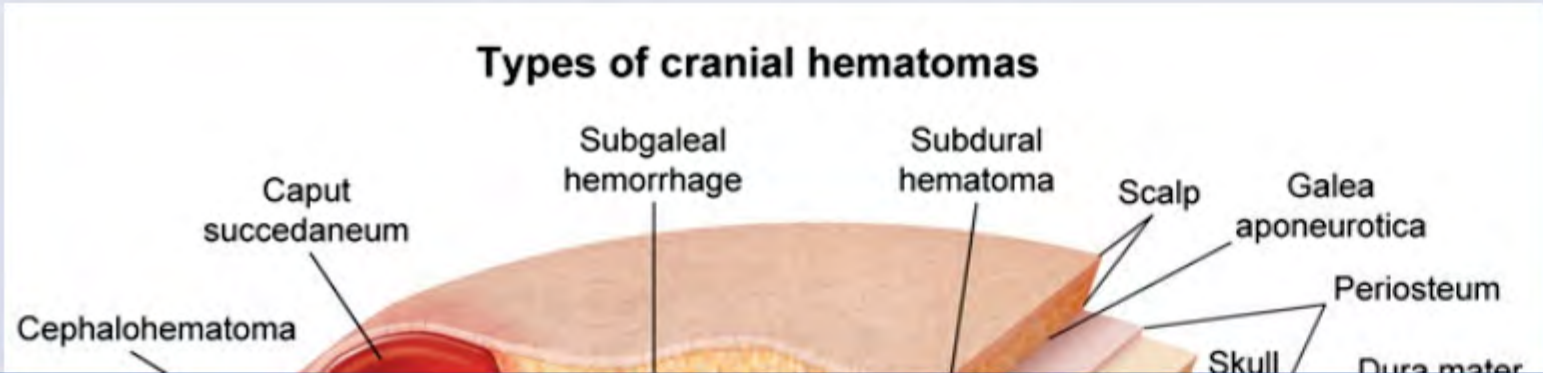
Correct answer
D

30%
Answered correctly

03 secs
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Explanation



A 10-minute-old boy is being evaluated in the delivery room. The patient was born at term to a 20-year-old primipara. The mother had a normal delivery. The newborn is alert and active. The physical examination reveals normal vital signs. The head circumference is 34 cm (95th percentile). The fontanelles are normal. The skull is normal. The scalp is normal. The face is normal. The chest is normal. The abdomen is normal. The extremities are normal. The newborn is being evaluated in the delivery room. The patient was born at term to a 20-year-old primipara. The mother had a normal delivery. The newborn is alert and active. The physical examination reveals normal vital signs. The head circumference is 34 cm (95th percentile). The fontanelles are normal. The skull is normal. The scalp is normal. The face is normal. The chest is normal. The abdomen is normal. The extremities are normal.

✗

✓

Ir

C

D

Ex

Exhibit Display

Types of cranial hematomas

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This newborn has diffuse, fluctuant swelling across the scalp and into the neck, findings concerning for a **subgaleal hemorrhage**.

Subgaleal hemorrhage is a rare, neonatal extracranial head injury caused by damage to the **emissary veins**, typically from **traction** on the scalp during vacuum-assisted delivery. Shearing of these veins, which connect veins of the scalp to the meningeal veins and dural sinuses, leads to hemorrhage in the potential space between the **periosteum** and **galea aponeurosis** (subgaleal space).

This potential space extends over the whole calvaria and into the neck. Therefore, **massive blood accumulation** (eg, 20%-40% of neonatal blood volume) can occur. Physical examination typically shows **diffuse, fluctuant scalp swelling** that extends **beyond suture lines** and potentially into the neck. The swelling may shift with movement and can continue to expand over 2-3 days. Progressive bleeding can lead to hypovolemia, shock, and death, making prompt recognition critical.

(Choices A, B, and C) Neonatal **intracranial bleeds** are also associated with assisted deliveries. Subarachnoid hemorrhage develops between the brain and arachnoid mater; subdural hematoma occurs between the arachnoid mater and dura mater; and epidural hematoma occurs between the dura mater and periosteum. Symptomatic intracranial bleeds typically present with seizures or hemodynamic instability, not diffuse scalp fluctuance.

(Choice E) Bleeding between the skull and periosteum results in a cephalohematoma, which usually presents as a small area of swelling over the parietal or occipital bone. Because the periosteum is fixed at suture lines (eg, sagittal, coronal), the firm, nonfluctuant swelling of a cephalohematoma does not cross suture lines and spread over the calvaria.

Educational objective:

Subgaleal hemorrhage is a potentially fatal neonatal bleed that occurs during delivery when emissary veins between the dural sinuses and scalp are sheared. The accumulation of blood between the periosteum and galea aponeurosis presents with a diffuse, progressive, fluctuant scalp and neck swelling; massive blood loss can lead to shock and death if not promptly recognized.

This newborn has diffuse, fluctuant swelling across the scalp and into the neck, findings concerning for a

sub

Exhibit Display

Types of intracranial hemorrhage

Epidural hematoma

Suture

Skull

Dura mater

Subdural hematoma

Dura mater

Arachnoid mater

Subarachnoid hemorrhage

Arachnoid mater

Pia mater

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shock and death if not promptly recognized.

A 37-year-old woman, gravida 2 para 1, at 28 weeks gestation comes to the office due to leakage of urine. The patient has had intermittent leakage with cough but no dysuria or hematuria. She reports normal fetal movement and has had an uncomplicated pregnancy. Four years ago, the patient had a spontaneous vaginal delivery of a 3500 g (7 lb 11 oz) neonate. The patient has no chronic medical conditions or prior surgeries. Vital signs are normal. Prepregnancy BMI was 32 kg/m². She has gained 15.8 kg (34.8 lb) during this pregnancy. The abdomen is gravid, and there is no suprapubic tenderness. Urinalysis is negative for blood, leukocyte esterase, and nitrite. Which of the following is the most likely mechanism for this patient's urinary incontinence?

- ☐ A. Bladder outlet obstruction
- ☐ B. Detrusor muscle hyperactivity
- ☐ C. Impaired detrusor contractility
- ☐ D. Increased intraabdominal pressure
- ☐ E. Sciatic nerve root compression
- ☐ F. Ureteral compression

Submit

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- A. Bladder outlet obstruction (1%)

B. Detrusor muscle hyperactivity (3%)

✗

C. Impaired detrusor contractility (5%)

✓

D. Increased intraabdominal pressure (84%)

E. Sciatic nerve root compression (0%)

F. Ureteral compression (3%)

Incorrect

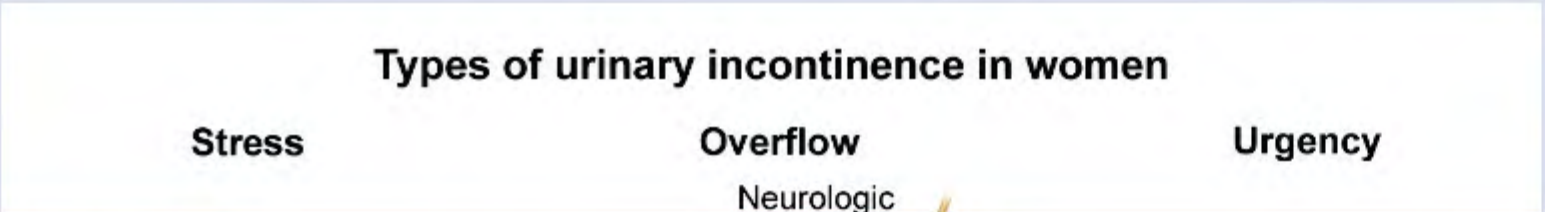
Correct answer
D

84%
Answered correctly

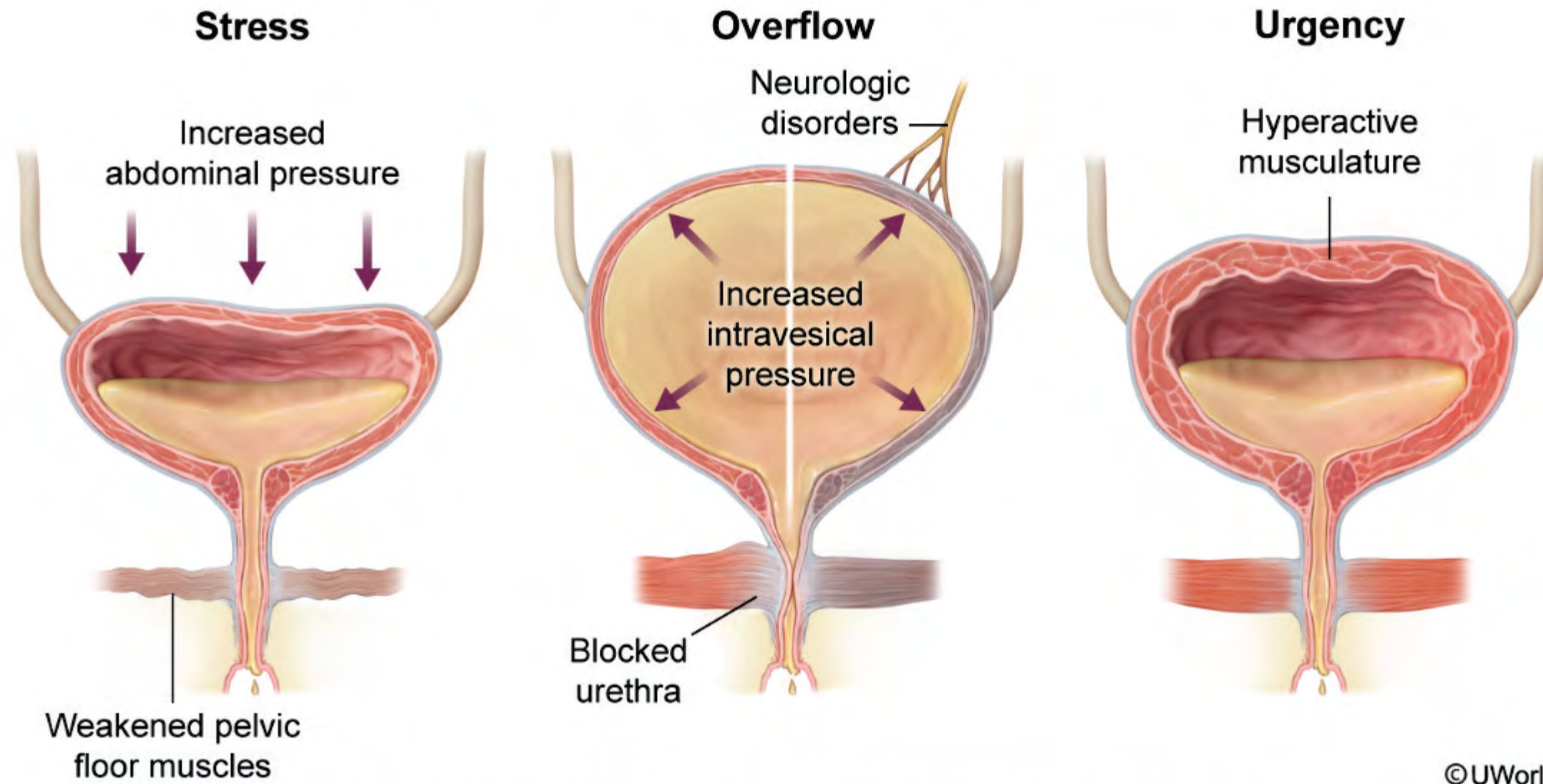
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Explanation



Types of urinary incontinence in women



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This patient with leakage of urine with coughing has **stress urinary incontinence** (SUI), a common condition during **pregnancy**. Risk factors include increasing parity, age, BMI, and maternal weight gain, as in this patient. Several physiologic changes in pregnancy contribute to SUI. The gravid uterus applies pressure on the bladder and stretches the connective tissue and muscles that normally support the pelvic organs. In addition, increased progesterone levels relax the muscles responsible for maintaining urinary continence: the external urethral sphincter and pelvic floor muscles (levator ani muscle complex). Normally, the external urethral sphincter compresses the urethra and creates a high urethral closing pressure. The pelvic floor muscles usually stabilize the urethra against the anterior vaginal wall and contract to decrease the **urethrovesical angle**, thereby kinking the urethra closed.

Because of **decreased urethral sphincter tone** and **pelvic floor muscle laxity**, the compression and position/angle of the urethra are compromised such that sudden **increases in intraabdominal pressure** (eg, **coughing**, sneezing) can cause the pressure within the bladder to exceed the urethral closing pressure. This leads to intermittent leakage of urine.

(Choices A and C) Bladder outlet obstruction (eg, urethral compression by uterine fibroids) and impaired detrusor contractility (eg, spinal cord injury) both lead to urinary retention. In patients with urinary retention, urine slowly accumulates in the bladder until the intravesical pressure exceeds the urethral closing pressure, causing urine leakage. Because urine constantly accumulates in the bladder, patients have persistent involuntary dribbling of urine (overflow incontinence) rather than intermittent leakage with cough.

(Choice B) Detrusor muscle hyperactivity is the mechanism behind urgency incontinence (overactive bladder syndrome). Patients typically have an intense urge to urinate followed by an immediate, involuntary loss of urine.

(Choice E) During pregnancy, the sciatic nerve can become compressed due to increased joint laxity from circulating relaxin and changes in maternal posture (eg, spinal lordosis). Sciatica commonly presents as lower back pain radiating down one side of the hip to the leg. It does not cause urinary incontinence.

(Choice F) During pregnancy, the gravid uterus compresses the ureters, leading to physiologic hydronephrosis.

sphincter and pelvic floor muscles (levator ani muscle complex). Normally, the external urethral sphincter compresses the urethra and creates a high urethral closing pressure. The pelvic floor muscles usually stabilize the urethra against the anterior vaginal wall and contract to decrease the **urethrovesical angle**, thereby kinking the urethra closed.

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(Choice F) During pregnancy, the gravid uterus compresses the ureters, leading to physiologic hydronephrosis and ureteral dilation. These changes increase the risk for urinary stasis and urinary tract infection but do not cause incontinence.

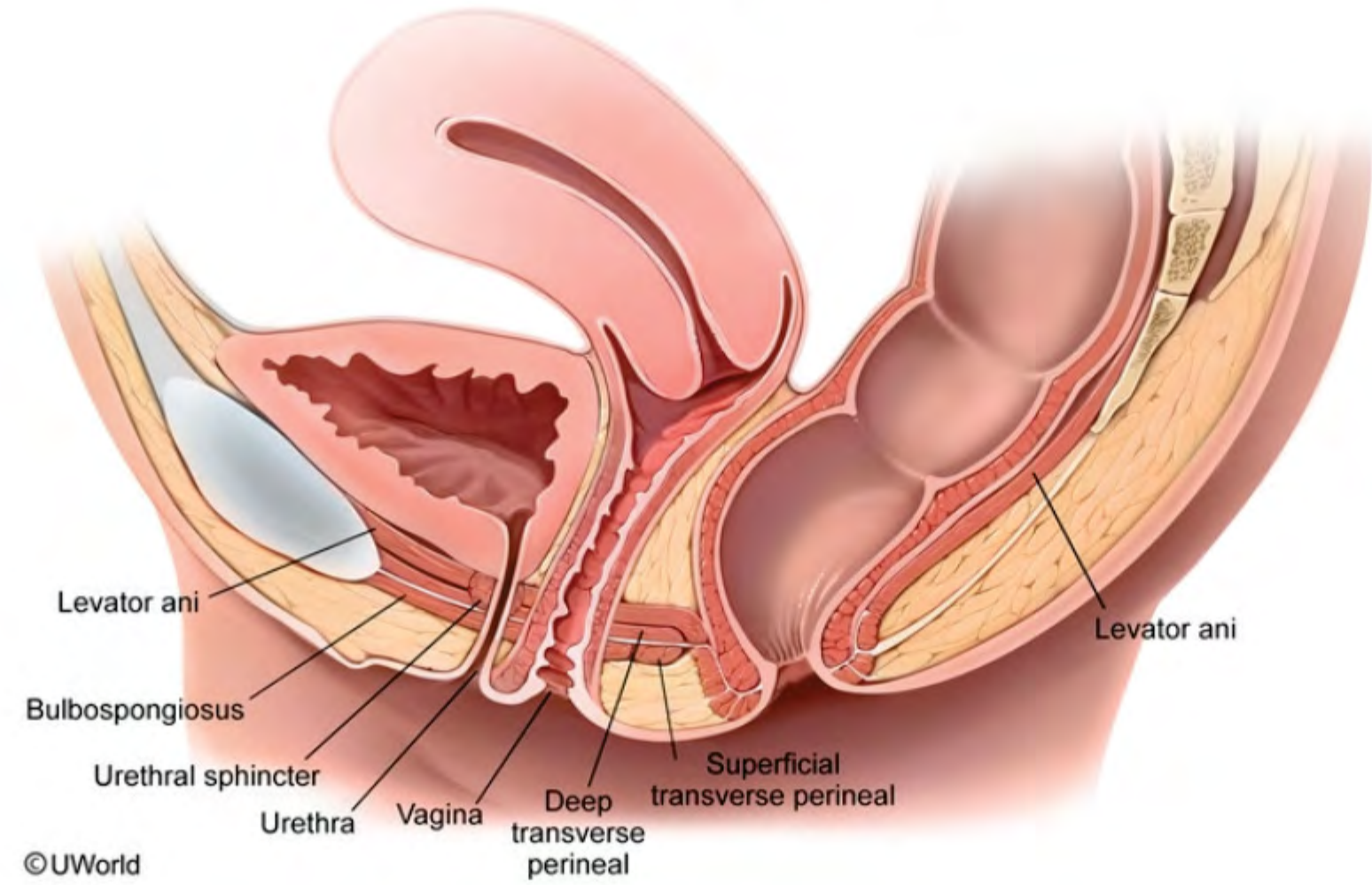
Educational objective:

Stress urinary incontinence, the leakage of urine with increased intraabdominal pressure (eg, coughing), is common in pregnancy due to decreased external urethral sphincter tone and increased pelvic floor muscle laxity.

Several physiologic changes in pregnancy contribute to SUI. The gravid uterus applies pressure on the bladder and stretches the connective tissue and muscles that normally support the pelvic organs. In addition, increased progesterone relaxes the smooth muscle of the ureters and the urethral sphincter.

Exhibit Display

Female pelvic floor muscles



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A day-old girl is being evaluated in the newborn nursery. The patient was born at 39 weeks gestation to a 38-year-old primigravida who immigrated to the United States during her third trimester. The patient had a strong cry and good tone at delivery but appears small for gestational age. Apgar scores were 8 and 9 at 1 and 5 minutes, respectively. Physical examination demonstrates white pupils bilaterally. A continuous harsh murmur is heard over the left infraclavicular area. The abdomen is nondistended, and there is no hepatosplenomegaly. Both ears fail the hearing screening. Which of the following maternal interventions would have most likely prevented this infant's condition?

- ☐ A. Maternal administration of a killed vaccine during pregnancy
- ☐ B. Maternal administration of a live attenuated vaccine before conception
- ☐ C. Maternal perinatal antibiotic treatment
- ☐ D. Maternal vitamin intake throughout pregnancy
- ☐ E. Viral enzyme inhibitor drug intake during the third trimester

Submit

A day-old girl is being evaluated in the newborn nursery. The patient was born at 39 weeks gestation to a 38-year-old primigravida who immigrated to the United States during her third trimester. The patient had a strong cry and good tone at delivery but appears small for gestational age. Apgar scores were 8 and 9 at 1 and 5 minutes, respectively. Physical examination demonstrates white pupils bilaterally. A continuous harsh murmur is heard over the left infraclavicular area. The abdomen is nondistended, and there is no hepatosplenomegaly. Both ears fail the hearing screening. Which of the following maternal interventions would have most likely prevented this infant's condition?

- ☐

A. Maternal administration of a killed vaccine during pregnancy (10%)
- ☒

B. Maternal administration of a live attenuated vaccine before conception (74%)
- ☐

C. Maternal perinatal antibiotic treatment (5%)
- ☐

D. Maternal vitamin intake throughout pregnancy (1%)
- ☐

E. Viral enzyme inhibitor drug intake during the third trimester (6%)

Correct

74%

Answered correctly

05 secs

Time Spent

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Version

Explanation

Congenital rubella syndrome	
Pathogenesis	<ul style="list-style-type: none">• Transplacental transmission
	<ul style="list-style-type: none">• Growth restriction

Congenital rubella syndrome	
Pathogenesis	<ul style="list-style-type: none">Transplacental transmission
Classic features	<ul style="list-style-type: none">Growth restrictionSensorineural hearing lossCongenital heart disease (eg, patent ductus arteriosus)Eye disease (eg, cataracts, glaucoma, retinopathy)CNS abnormalities (eg, developmental delay)
Diagnosis	<ul style="list-style-type: none">Rubella IgMPCR or viral culture
Prevention	<ul style="list-style-type: none">Maternal preconception immunization with live attenuated rubella vaccine

This neonate has the classic triad of findings for **congenital rubella syndrome** (CRS), including **cataracts** (white pupils), **sensorineural hearing loss**, and **patent ductus arteriosus** (continuous, harsh murmur). Growth restriction and CNS involvement may also occur. CRS is caused by transplacental transmission of the rubella virus from an infected mother. In pregnant women, rubella infection can cause nonspecific flu-like symptoms (eg, fever, malaise), arthralgias, and a rash with cephalocaudal progression. However, infection can also be asymptomatic, which likely occurred in this case.

Prevention of CRS is achieved by preventing maternal illness. Maternal rubella infection is rare in the United States because of universal childhood vaccination with the measles, mumps, and rubella (MMR) vaccine. However, lack of immunization programs in certain areas (eg, Southeast Asia, Africa) is associated with higher rates of CRS.

In contrast to killed vaccines that induce only humoral immunity, the rubella vaccine is a **live attenuated vaccine** and therefore induces both humoral and cell-mediated immunity (**Choice A**). Vaccination should be administered

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In contrast to killed vaccines that induce only humoral immunity, the rubella vaccine is a **live attenuated vaccine** and therefore induces both humoral and cell-mediated immunity (**Choice A**). Vaccination should be administered **prior to conception** because live attenuated vaccines carry the potential risk of viral transmission to the fetus if given during pregnancy.

(Choice C) Maternal perinatal antibiotic treatment is indicated for chorioamnionitis and group B *Streptococcus* colonization, which are risk factors for neonatal sepsis. Signs of sepsis in the immediate newborn period can include lethargy, temperature instability, and/or respiratory distress, not cataracts and hearing loss.

(Choice D) Prenatal folic acid helps prevent neural tube defects, in which the brain and/or spinal cord fail to develop properly. Affected infants may have a **cutaneous abnormality** (eg, hair tuft) in the lumbosacral area, lower extremity weakness, or hypotonia.

(Choice E) Antiviral therapy is indicated for pregnant women with HIV to decrease the risk of vertical transmission. In contrast to this patient, newborns with HIV are typically asymptomatic and develop failure to thrive and chronic diarrhea later in infancy.

Educational objective:

from an infected mother. In pregnant women, rubella infection can cause nonspecific flu-like symptoms (eg, fever, malaise), arthralgias, and a rash with cephalocaudal progression. However, infection can also be asymptomatic, which likely occurred in this case.

Prevention of CRS is achieved by preventing maternal illness. Maternal rubella infection is rare in the United States because of universal childhood vaccination with the measles, mumps, and rubella (MMR) vaccine. However, lack of immunization programs in certain areas (eg, Southeast Asia, Africa) is associated with higher rates of CRS.

In contrast to killed vaccines that induce only humoral immunity, the rubella vaccine is a **live attenuated vaccine** and therefore induces both humoral and cell-mediated immunity (**Choice A**). Vaccination should be administered **prior to conception** because live attenuated vaccines carry the potential risk of viral transmission to the fetus if given during pregnancy.

(Choice C) Maternal perinatal antibiotic treatment is indicated for chorioamnionitis and group B *Streptococcus* colonization, which are risk factors for neonatal sepsis. Signs of sepsis in the immediate newborn period can include lethargy, temperature instability, and/or respiratory distress, not cataracts and hearing loss.

(Choice D) Prenatal folic acid helps prevent neural tube defects, in which the brain and/or spinal cord fail to develop properly. Affected infants may have a **cutaneous abnormality** (eg, hair tuft) in the lumbosacral area, lower extremity weakness, or hypotonia.

(Choice E) Antiviral therapy is indicated for pregnant women with HIV to decrease the risk of vertical transmission. In contrast to this patient, newborns with HIV are typically asymptomatic and develop failure to thrive and chronic diarrhea later in infancy.

Educational objective:

The classic triad of findings for congenital rubella syndrome includes cataracts, sensorineural hearing loss, and patent ductus arteriosus. Prevention consists of maternal preconception immunization with the live attenuated rubella vaccine.

from an infected mother. In pregnant women, rubella infection can cause nonspecific flu-like symptoms (eg, fever, malaise), arthralgias, and a rash with cephalocaudal progression. However, infection can also be asymptomatic, which

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Exhibit Display

Common dermatologic manifestations of closed spinal dysraphism (spina bifida occulta)

Spinal process defect



Dimple (>5 mm long & >2.5 cm above anus)



Hair tuft



Hemangioma



Subcutaneous mass (eg, lipoma)



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A healthy, nulliparous 29-year-old woman with regular 28-day cycles comes to the office for a fertility evaluation. Over the past year, she and her 30-year-old husband have been trying to conceive. Her only medications are prenatal vitamins. She tracks her periods, cervical mucus, basal temperature, and intercourse with a smartphone app. The couple has intercourse every other day from cycle day 8 through the LH surge. The patient's urine pregnancy test is positive. The oocyte was most likely arrested in which of the following stages of meiosis immediately prior to fertilization?

- ☐ A. Metaphase of meiosis I
- ☐ B. Metaphase of meiosis II
- ☐ C. Prophase of meiosis I
- ☐ D. Prophase of meiosis II
- ☐ E. Telophase of meiosis I
- ☐ F. Telophase of meiosis II

Submit

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- ☐

A. Metaphase of meiosis I (6%)
- ☒

B. Metaphase of meiosis II (62%)
- ☐

C. Prophase of meiosis I (11%)
- ☐

D. Prophase of meiosis II (16%)
- ☐

E. Telophase of meiosis I (1%)
- ☐

F. Telophase of meiosis II (1%)

Correct

62%

Answered correctly

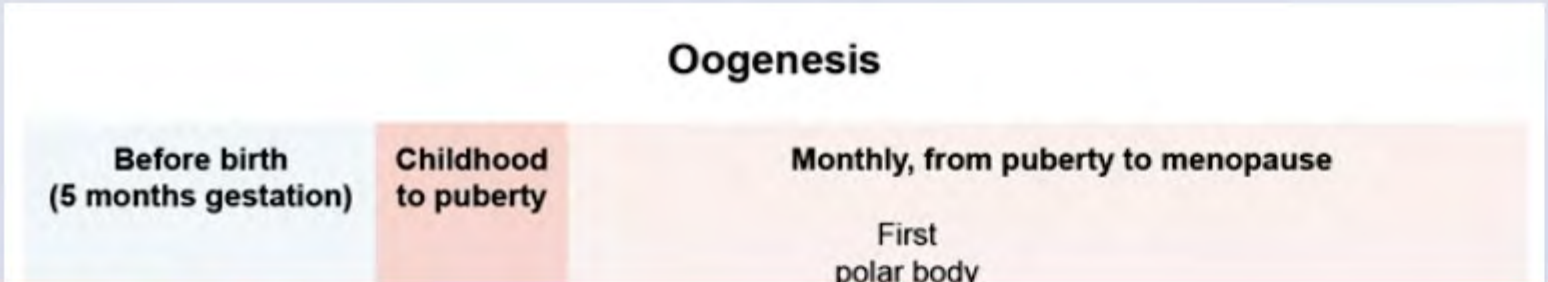
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Explanation



A healthy nulliparous 29-year-old woman with regular 28-day cycles comes to the office for a fertility evaluation.

Over the past year, she has been unable to conceive. She has a history of premenstrual syndrome, which is managed with over-the-counter pain relievers. She is currently not pregnant and has no other medical conditions.

Exhibit Display

Oogenesis

The diagram illustrates the process of oogenesis, divided into three main phases:

- Before birth (5 months gestation):** An **Oogonium** undergoes **Mitosis** to become a **Primary oocyte**.
- Childhood to puberty:** The **Primary oocyte** remains in **Meiosis I arrested in prophase**.
- Monthly, from puberty to menopause:**
 - The **Primary oocyte** completes **Meiosis I**, resulting in a **Secondary oocyte** and a **First polar body**. The **First polar body** **Degenerates**.
 - The **Secondary oocyte** undergoes **Ovulation** and enters **Meiosis II arrested in metaphase**.
 - Upon fertilization, **Meiosis II completed when fertilized**, resulting in an **Ovum** and a **Polar body**.
 - The **Ovum** and **Sperm** fuse to form a **Zygote**, which contains **Pronuclei** and **Polar bodies**.

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Female gametogenesis begins in utero at approximately 4 weeks gestation when primordial germ cells migrate from the yolk sac region to the developing gonadal region. These germ cells then differentiate into **oogonia** and multiply by mitosis before beginning meiosis I. Now called **primary oocytes**, these cells arrest in **prophase of meiosis I (Choice C)** and remain there until ovulation. The chromosome pairs are arranged in a tetrad during this phase, and their proximity allows for genetic recombination. The full complement of oocytes is developed by 5 months gestation and then degenerates during the woman's lifetime until depleted at menopause.

At puberty, ovulatory cycles begin and the female is capable of reproduction. During each menstrual cycle, FSH stimulation followed by an LH surge causes some oocytes to resume meiosis I. Telophase I (**Choice E**) is the last phase of meiosis I and yields secondary oocytes and polar bodies. The secondary oocyte begins meiosis II (the polar body degenerates) but halts in metaphase II. Approximately 36 hours after LH levels begin to rise, a secondary oocyte is released from the ovarian follicle. The secondary oocyte remains frozen in **metaphase II until fertilization occurs**, at which point it completes its division (telophase II, **Choice F**) into a mature oocyte (ovum) and second polar body. A normally fertilized embryo is then diploid due to genetic contribution from both the ovum and sperm pronuclei.





(Choice A) Metaphase of meiosis I differs from metaphase of meiosis II and mitosis, as the spindles are not preparing to split the centromeres. Instead, the tetrads are lined up along the metaphase plate, and during anaphase the homologs separate from each other with the centromeres intact.

(Choice D) Prophase of meiosis II is unique as it does not immediately follow a period of DNA synthesis; DNA was duplicated prior to prophase of meiosis I.

Educational objective:

Primary oocytes are completely developed in female fetuses by the fifth month of gestation, at which point they are arrested in prophase of meiosis I. After puberty, menstrual cycle hormones stimulate the primary oocyte to resume differentiation. Prior to fertilization, secondary oocytes are arrested in metaphase of meiosis II.

Exhibit Display

Gametogenesis					
Female	Male	Cell cycle stage	# homologs (n)	# chromatids (c)	
Oogonia	Spermatagonia	G0	2n (46)	2c	
Primary oocyte	Primary spermatocyte	Prophase MI	2n (46)	4c	
Secondary oocyte	Secondary spermatocyte	Metaphase MII	1n (23)	2c	
Mature ovum	Spermatid	Telophase MII	1n (23)	1c	

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A 28-year-old woman, gravida 1 para 0, at 16 weeks gestation comes to the office for a prenatal visit. The patient feels well and has no concerns. Medical history is significant for recurrent urinary tract infections. Her only medication is a prenatal multivitamin. Blood pressure is 116/68 mm Hg and pulse is 80/min. Physical examination shows no abnormalities and fetal heart tones are normal. Urine dipstick reveals no blood or leukocyte esterase but is positive for protein. Laboratory testing shows the following:

Serum chemistry		
Sodium		138 mEq/L
Potassium		4.2 mEq/L
Bicarbonate		24 mEq/L
Creatinine		0.6 mg/dL
Glucose		80 mg/dL
Liver function studies		
Albumin		4.5 g/dL
Total bilirubin		0.8 mg/dL
Alkaline phosphatase		60 U/L
Aspartate aminotransferase (SGOT)		22 U/L
Alanine aminotransferase (SGPT)		24 U/L
Urinalysis		1+ protein; no cells or casts

A 24-hour urinary protein excretion is 200 mg. Which of the following processes is most likely contributing to this patient's urinary findings?

Bicarbonate	24 mEq/L
Creatinine	0.6 mg/dL
Glucose	80 mg/dL
Liver function studies	
Albumin	4.5 g/dL
Total bilirubin	0.8 mg/dL
Alkaline phosphatase	60 U/L
Aspartate aminotransferase (SGOT)	22 U/L
Alanine aminotransferase (SGPT)	24 U/L
Urinalysis	1+ protein; no cells or casts

A 24-hour urinary protein excretion is 200 mg. Which of the following processes is most likely contributing to this patient's urinary findings?

- ☐ A. Effacement of podocyte foot processes

☐ B. Generalized endothelial dysfunction

☐ C. Increased glomerular filtration rate

☐ D. Increased mesangial extracellular matrix

☐ E. Shedding of injured tubular epithelial cells

Submit

Bicarbonate	24 mEq/L
Creatinine	0.6 mg/dL
Glucose	80 mg/dL
Liver function studies	
Albumin	4.5 g/dL
Total bilirubin	0.8 mg/dL
Alkaline phosphatase	60 U/L
Aspartate aminotransferase (SGOT)	22 U/L
Alanine aminotransferase (SGPT)	24 U/L
Urinalysis	1+ protein; no cells or casts

A 24-hour urinary protein excretion is 200 mg. Which of the following processes is most likely contributing to this patient's urinary findings?

-
-

Incorrect

Correct answer
C

69%

Answered correctly

10 secs

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Exhibit Display

Renal adaptations during pregnancy

The diagram illustrates the physiological changes in the kidneys during pregnancy. It shows a cross-section of the kidney with the renal cortex and medulla. The renal artery and vein are shown entering the kidney. A box on the left lists the factors contributing to the changes: Relaxin-induced arteriolar dilation and decreased serum albumin. These factors lead to an increase in renal plasma flow, indicated by a red arrow pointing into the kidney. This increase in flow leads to an increase in glomerular filtration rate (GFR) and creatinine clearance, indicated by a green arrow pointing down from the glomerulus. The diagram also shows the glomerular capillaries with an increase in capillary hydrostatic pressure and a decrease in capillary oncotic pressure, which further contributes to the increased GFR.

- Relaxin-induced arteriolar dilation
- ↓ serum albumin

↑ renal plasma flow

↑ capillary hydrostatic pressure
↓ capillary oncotic pressure

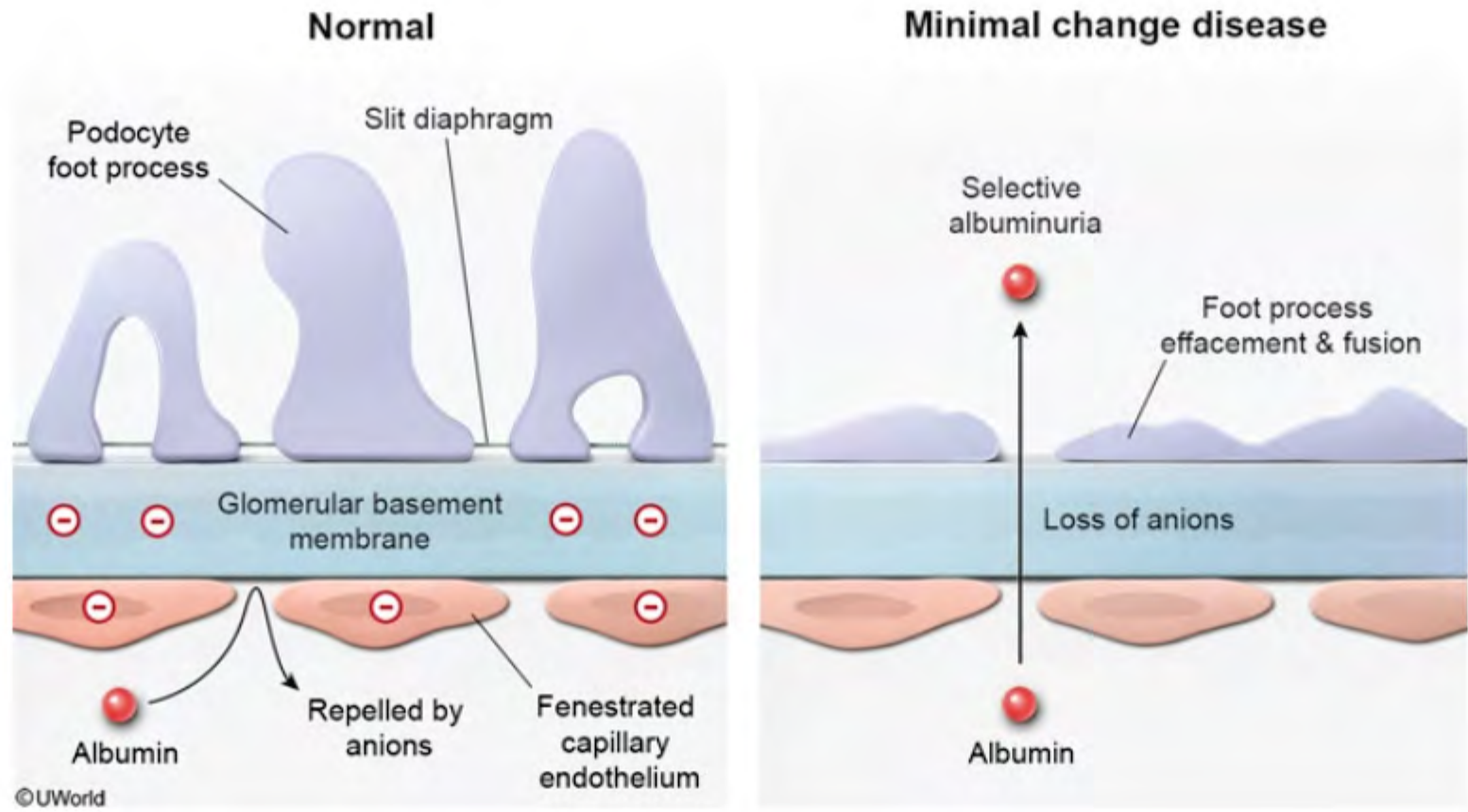
Increased GFR
Increased creatinine clearance

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excretion (ie, <300 mg/24 hr) is a normal finding in pregnancy.

A 37-year-old woman, gravida 2 para 1, at 30 weeks gestation comes to the office to discuss her recent laboratory work. At her last visit at 28 weeks gestation, the patient drank a solution containing 50 g of glucose, and her serum glucose was abnormally elevated an hour later. Today, she is scheduled to undergo a confirmatory 3-hour glucose test. Her pregnancy has been uncomplicated, and the patient has no chronic medical conditions. She is taking a daily prenatal vitamin. Vital signs are normal. Physical examination shows a uterine size consistent with gestational age. Fetal heart rate is 140/min. Which of the following hormones is most likely contributing to this patient's abnormal laboratory findings?

- ☐ A. Growth hormone
- ☐ B. Human chorionic gonadotropin
- ☐ C. Human placental lactogen
- ☐ D. Prolactin
- ☐ E. Thyroxine

Submit

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- A. Growth hormone (14%)

✖

B. Human chorionic gonadotropin (9%)

✔

C. Human placental lactogen (70%)

D. Prolactin (2%)

E. Thyroxine (3%)

Incorrect

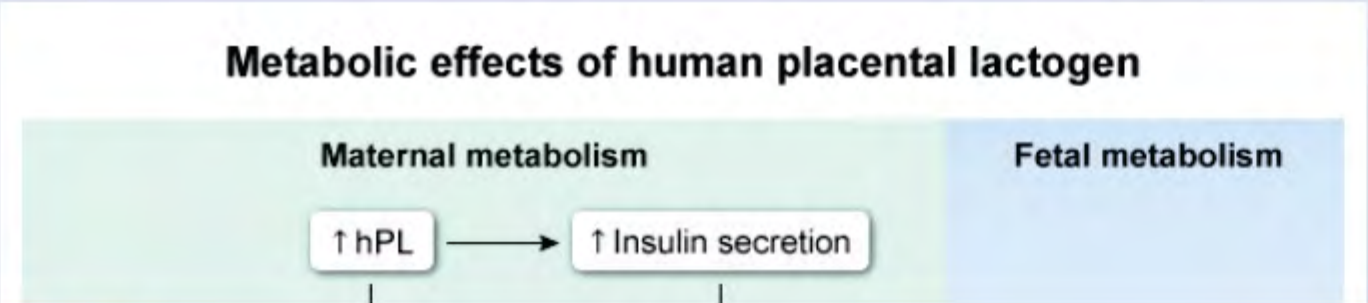
Correct answer
C

70%
Answered correctly

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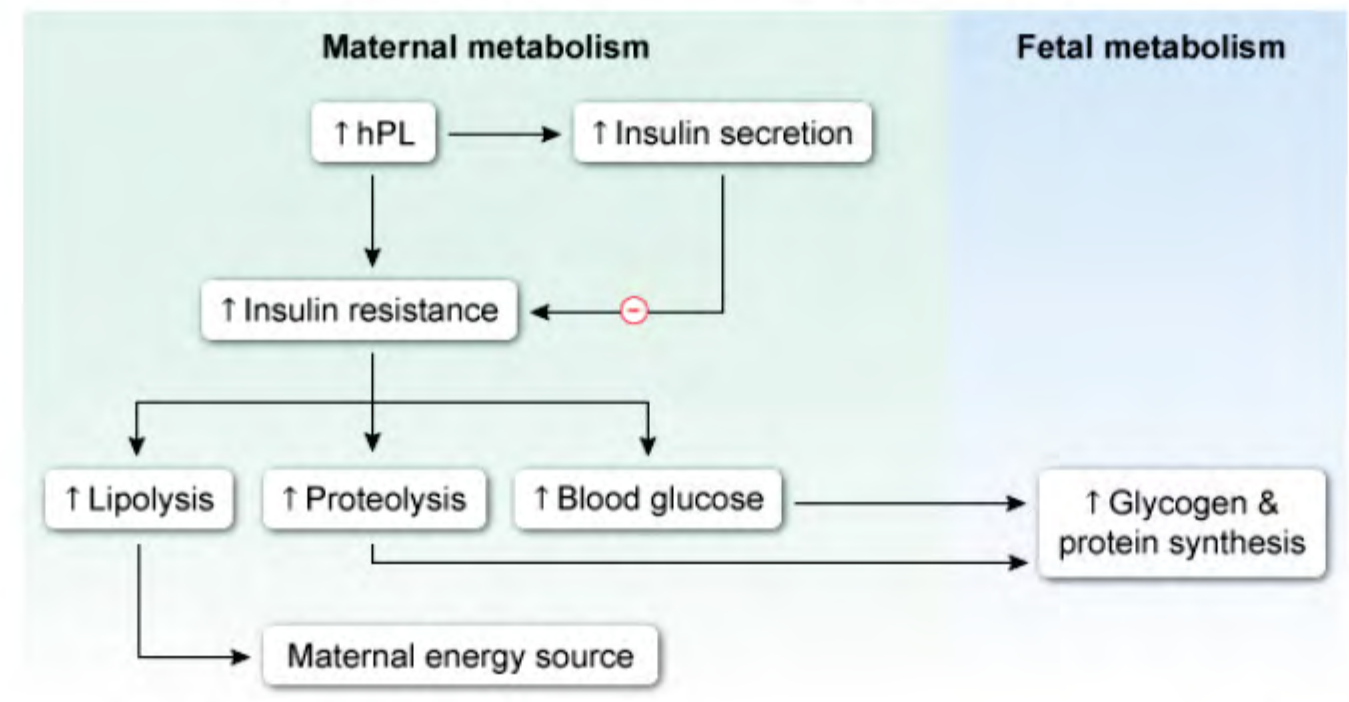
Explanation



B. Human chorionic gonadotropin (9%)

Exhibit Display

Metabolic effects of human placental lactogen



hPL = human placental lactogen.

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hPL = human placental lactogen.

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During pregnancy, a physiologic increase in maternal **insulin resistance** occurs during the second and third trimesters, largely due to **human placental lactogen** (hPL), a peptide hormone secreted by the syncytiotrophoblast. hPL establishes a glucose-sparing effect that results in:

- **Increased maternal glucose levels:** allows glucose to freely cross the placenta for consumption by the fetus for energy
- **Increased maternal proteolysis:** provides a readily available supply of amino acids for the fetus
- **Increased maternal lipolysis:** leads to increased free fatty acids and ketones to provide energy to the mother, preserving glucose and amino acids for the fetus

hPL also stimulates maternal **pancreatic beta-cell proliferation** to increase insulin production to counteract the rising resistance. This physiologic process becomes pathologic (ie, **gestational diabetes mellitus**) when the pancreatic function is not sufficient to overcome the pregnancy-related increase in insulin resistance.

Average levels of hPL rise with increasing gestational age, reflecting the increasing energy requirements of the growing fetus. Therefore, screening for gestational diabetes is performed in the late second or early third trimester (when plasma levels of hPL are peaking) to ensure screening accuracy.

(Choice A) Growth hormone, released from the pituitary gland, causes an increase in insulin resistance and fat breakdown; however, growth hormone secretion does not increase during pregnancy.

(Choice B) In early pregnancy, the placental **syncytiotrophoblast** secretes human chorionic gonadotropin, a peptide hormone, which stimulates the corpus luteum to produce progesterone, a steroid hormone. Progesterone is necessary to maintain the secretory endometrium, allowing the pregnancy to persist through term.

(Choice D) Prolactin is a peptide hormone secreted by lactotroph cells in the anterior pituitary. It stimulates ductal and alveolar growth in breast tissue and is responsible for milk production and lactation in postpartum women.

(Choice E) Pregnancy increases the levels of thyroid-binding globulin, leading to increased total circulating T₃ and T₄. However, free thyroid hormone levels remain essentially normal.

- **Increased maternal proteolysis:** provides a readily available supply of amino acids for the fetus
- **Increased maternal lipolysis:** leads to increased free fatty acids and ketones to provide energy to the mother, preserving glucose and amino acids for the fetus

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(Choice E) Pregnancy increases the levels of thyroid-binding globulin, leading to increased total circulating T₃ and T₄. However, free thyroid hormone levels remain essentially normal.

Educational objective:

Human placental lactogen creates a fetal glucose-sparing effect by increasing maternal insulin resistance during the second and third trimesters, leading to a rise in serum glucose that helps provide adequate nutrition to the growing fetus. Gestational diabetes occurs when the compensatory rise in maternal insulin secretion is inadequate to prevent serum glucose levels from reaching excessively high levels.

hPL also stimulates maternal **pancreatic beta-cell proliferation** to increase insulin production to counteract the

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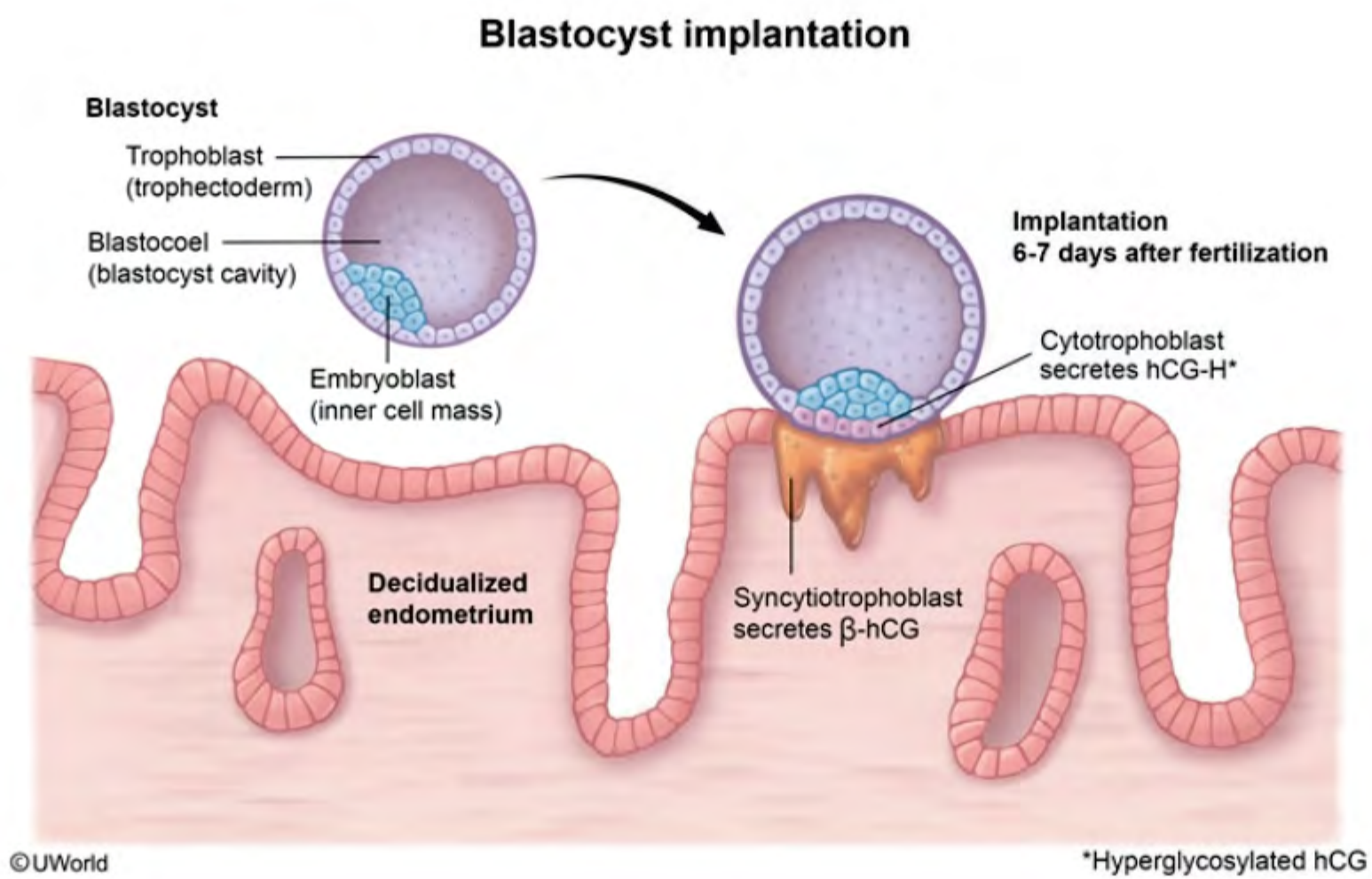
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A 29-year-old woman, gravida 1 para 0, at 28 weeks gestation comes to the office to establish prenatal care after recently moving to a new city. She has had no complications in this pregnancy and had a normal anatomy ultrasound at 20 weeks gestation. On physical examination, the fundal height measures 28 cm. Cardiac auscultation reveals a 2/6 blowing systolic murmur best heard at the left sternal border. The murmur intensifies with inspiration, and there is no ejection click or palpable thrill at the sternal notch. The lungs are clear to auscultation. There is 1+ pitting edema to the midshins bilaterally. Which of the following is most likely responsible for this patient's murmur?

- ☐ A. Bacterial vegetations on the mitral valve
- ☐ B. Calcification of a bicuspid aortic valve
- ☐ C. High blood flow across the pulmonic valve
- ☐ D. Inflammatory stiffening of the tricuspid valve
- ☐ E. Myxomatous degeneration of the mitral valve

Submit

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- A. Bacterial vegetations on the mitral valve (2%)

✖

B. Calcification of a bicuspid aortic valve (3%)

C. High blood flow across the pulmonic valve (79%)

D. Inflammatory stiffening of the tricuspid valve (9%)

E. Myxomatous degeneration of the mitral valve (4%)

Incorrect

Correct answer
C

79%

Answered correctly

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Explanation

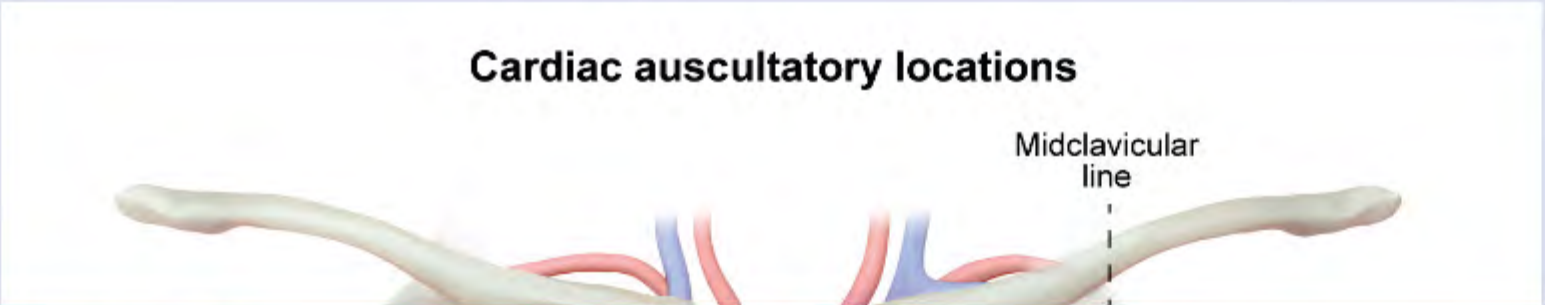
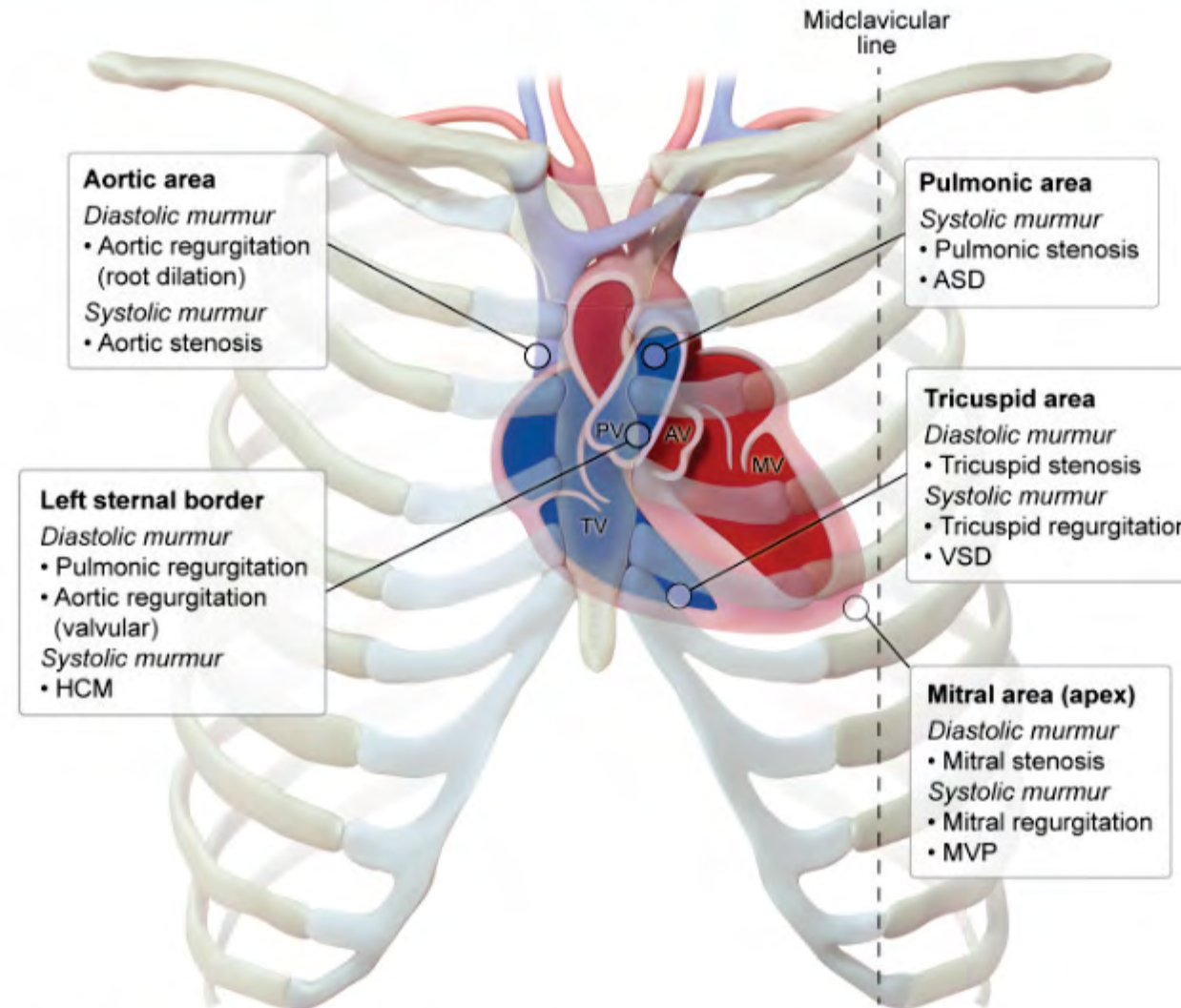


Exhibit Display

Cardiac auscultatory locations



ASD = atrial septal defect; AV = aortic valve; HCM = hypertrophic cardiomyopathy; MV = mitral valve; MVP = mitral valve prolapse; PV = pulmonic valve; TV = tricuspid valve; VSD = ventricular septal defect.
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Beginning at 6 weeks gestation, **maternal blood volume** begins to **increase** to help accommodate the developing fetus; this increase can reach up to 50% from baseline toward the end of the third trimester. The cardiovascular system adapts accordingly with ventricular dilation and **increased cardiac output**. These changes can cause several auscultation findings during **normal pregnancy**:

- **Pulmonic flow murmur:** This is a low-grade systolic murmur best heard at the left upper or midsternal border. It is caused by **high blood flow** across the pulmonic valve. The anterior location of the pulmonic valve helps make the murmur relatively easily detectable. Like most right-sided murmurs, the intensity increases during inspiration as additional blood is pulled into the lungs and the right side of the heart.
- **S3:** This is caused by reverberation of sound during diastolic filling of an enlarged left ventricle. It is commonly associated with heart failure but is considered a normal finding in pregnancy.
- **Venous hum:** This is a continuous murmur likely caused by brisk blood flow through the jugular veins, and it is usually best heard in the right supraclavicular fossa. Overall, it is rare in adults but is commonly present in high-flow states such as pregnancy.

(Choice A) Bacterial vegetation on the mitral valve can occur with infective endocarditis, typically leading to mitral regurgitation. The murmur of mitral regurgitation is a holosystolic murmur best heard at the cardiac apex, with decreased intensity during inspiration.

(Choice B) Calcification of a congenital bicuspid aortic valve is a common cause of aortic stenosis. Affected patients are typically age >40 and have a **crescendo-decrescendo** systolic murmur best heard at the right upper sternal border. Like most left-sided murmurs, the murmur tends to decrease in intensity with inspiration.

(Choice D) Inflammatory stiffening of the tricuspid valve can occur with rheumatic heart disease, leading to tricuspid stenosis. The murmur of tricuspid stenosis increases in intensity with inspiration, but it is diastolic rather than systolic.

(Choice E) Myxomatous degeneration of the mitral valve can lead to mitral valve prolapse, which is recognized by

valve helps make the murmur relatively easily detectable. Like most right-sided murmurs, the intensity increases during inspiration as additional blood is pulled into the lungs and the right side of the heart.

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(Choice E) Myxomatous degeneration of the mitral valve can lead to mitral valve prolapse, which is recognized by a midsystolic click that may be followed by a murmur of mitral regurgitation. The timing (but not the intensity) of the click varies with changes in left ventricular blood volume.

Educational objective:

Normal pregnancy is associated with an increase in blood volume and cardiac output that frequently causes a pulmonic flow murmur and S3.

valve helps make the murmur relatively easily detectable. Like most right-sided murmurs, the intensity

Exhibit Display

Cardiac murmurs

Systolic	Aortic stenosis*		Crescendo-decrescendo at RUSB, A2 soft & delayed
	Mitral regurgitation*		Holosystolic at apex & radiates to axilla
	Hypertrophic cardiomyopathy		Crescendo-decrescendo at mid-left sternal border
	Mitral valve prolapse		Midsystolic click followed by late systolic murmur
	Atrial septal defect		Midsystolic with wide & fixed splitting of S2
	Ventricular septal defect		Holosystolic at left sternal border
Diastolic	Aortic regurgitation*		Decrescendo at LUSB (valvular), RUSB (aortic root)
	Mitral stenosis*		Opening snap followed by middiastolic rumble with presystolic accentuation
Continuous	Patent ductus arteriosus		Machinery-like at LUSB or left infraclavicular area

*Pulmonic & tricuspid valve murmurs are similar in character to their aortic & mitral valve counterparts but with different location of optimal auscultation. These murmurs can be further differentiated from their left-sided counterparts by an increase in intensity with inspiration.
LUSB = left upper sternal border; RUSB = right upper sternal border.
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A newborn boy is in the neonatal intensive care unit for mechanical ventilation due to pulmonary hypoplasia. He was born via spontaneous vaginal delivery at 30 weeks gestation to a 41-year-old woman who did not receive prenatal care. The mother had assumed her amenorrhea was due to early menopause, which runs in her family. She took multiple medications for poorly controlled hypertension before realizing she was pregnant. Prenatal ultrasound demonstrated severe oligohydramnios for which delivery was induced. Examination of the infant shows an intubated and sedated boy whose weight is at the third percentile. The temporal, occipital, and parietal bones are underdeveloped, and the right lower limb is shortened and contracted. Which of the following mechanisms is the most likely cause of this neonate's abnormalities?

- ☐ A. Alpha-2 adrenergic agonism
- ☐ B. Beta-adrenergic receptor blockade
- ☐ C. Cellular damage from free radicals
- ☐ D. Impaired metabolism of angiotensin I
- ☐ E. Impaired oxygen delivery
- ☐ F. Impaired prostaglandin metabolism

Submit

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- A. Alpha-2 adrenergic agonism (1%)

✖

B. Beta-adrenergic receptor blockade (7%)

C. Cellular damage from free radicals (4%)

✔

D. Impaired metabolism of angiotensin I (68%)

E. Impaired oxygen delivery (12%)

F. Impaired prostaglandin metabolism (5%)

Incorrect

Correct answer
D

68%
Answered correctly

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Time Spent

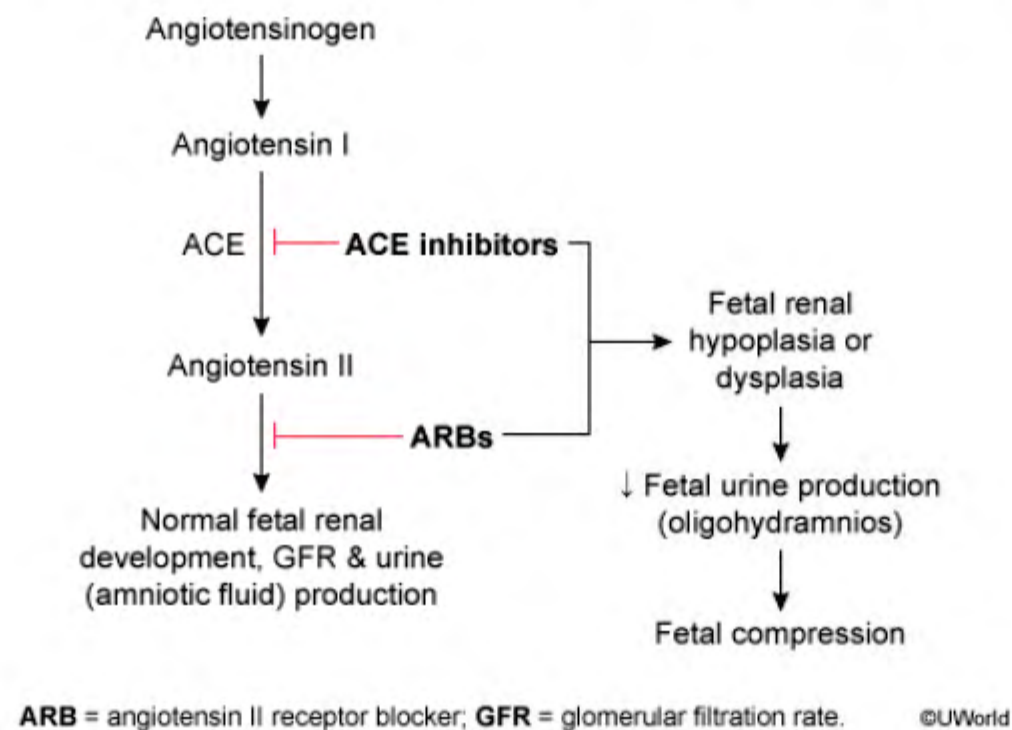
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Explanation

Teratogenic effects of ACE inhibitors and ARBs

Angiotensinogen

Teratogenic effects of ACE inhibitors and ARBs



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- **ACE inhibitors** impair metabolism of angiotensin I to angiotensin II by inhibiting ACE activity, thereby

This neonate's abnormalities are likely due to exposure to an ACE inhibitor and/or angiotensin II receptor blocker (ARB), which are highly teratogenic antihypertensive medications. During pregnancy, ACE inhibitors and ARBs can cross the placenta and decrease angiotensin II activity:

- **ACE inhibitors impair metabolism of angiotensin I** to angiotensin II by inhibiting ACE activity, thereby decreasing angiotensin II levels.
- **ARBs block the angiotensin II receptor** type 1 (AT1), which decreases angiotensin II activity.

The decrease in angiotensin II causes **abnormal fetal renal development** (eg, renal hypoplasia) and a reduced fetal glomerular filtration rate (GFR). Hypoplastic kidneys with reduced GFR produce low volumes of fetal urine, which, as the main component of amniotic fluid, can lead to **oligohydramnios**.

Because amniotic fluid enables fetal lung development and protects against limb compression, neonates with severe oligohydramnios are at risk for **pulmonary hypoplasia** and **limb defects** (ie, [Potter sequence](#)). This neonate also has hypocalvaria (eg, temporal, occipital, and parietal bone hypoplasia) due to impaired cranial vascularization and severe growth restriction (likely due to both fetal compression and maternal hypertension).

(Choices A and B) Methyldopa (alpha-2 adrenergic agonist), labetalol (beta-adrenergic antagonist), and nifedipine (calcium channel blocker) are the preferred antihypertensive medications during pregnancy. Although they can cause neonatal adverse effects (eg, hypotension [methyldopa], hypoglycemia [labetalol]), none are associated with abnormal fetal renal development or oligohydramnios.

(Choice C) Radiation causes cellular damage due to molecular bond disruption by free radicals. Fetal radiation exposure increases the risk for cancer, particularly childhood leukemia.

(Choice E) Impaired fetal oxygen delivery can occur with maternal anemia, tobacco use, or placental insufficiency (in which there is decreased placental perfusion [eg, preeclampsia, fetal chromosomal abnormalities]). Although decreased oxygenation can lead to fetal growth restriction, it does not typically cause oligohydramnios and limb deformity.

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(Choice F) Nonsteroidal anti-inflammatory drugs decrease prostaglandin E₁ production, causing vasoconstriction. Although the fetal GFR may decrease as a result, the associated oligohydramnios is typically mild and transient. In contrast, ACE inhibitors and ARBs cause long-lasting abnormalities in renal development.

Educational objective:

ACE inhibitors and angiotensin II receptor blockers are teratogens. Both decrease angiotensin II activity, leading to abnormal fetal renal development, oligohydramnios, and potential neonatal Potter sequence (ie, pulmonary hypoplasia, limb deformity).

The decrease in angiotensin II causes **abnormal fetal renal development** (eg, renal hypoplasia) and a reduced fetal glomerular filtration rate (GFR). Hypoplastic kidneys with reduced GFR produce low volumes of fetal urine, which

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Exhibit Display

Potter sequence

```
graph TD; A["Urinary tract anomaly  
(eg, bilateral renal  
agenesis) or PPRM  
at <26 weeks"] --> B["Anuria/oliguria in utero"]; B --> C["Severe  
oligohydramnios  
(↑ fetal compression)"]; C --> D["Flat facies"]; C --> E["Pulmonary hypoplasia"]; C --> F["Limb deformities"];
```

Urinary tract anomaly (eg, bilateral renal agenesis) or PPRM at <26 weeks

Anuria/oliguria in utero

Severe oligohydramnios (↑ fetal compression)

Flat facies

Pulmonary hypoplasia

Limb deformities

PPROM = preterm prelabor rupture of membranes.

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A 38-year-old woman, gravida 1 para 0, at 34 weeks gestation comes to the office due to increasing swelling in her hands and legs. The patient first noticed the swelling a few weeks ago but now cannot stand for more than an hour without needing to elevate her legs. She has no orthopnea, dyspnea, or chest pain. The patient has no chronic medical conditions and takes no daily medications. Vital signs are normal. Cardiac examination is within normal limits. Lungs are clear to auscultation. Bilateral lower extremities have 2+ pitting edema to the midshin. Urine dipstick shows no protein. Compared to a healthy nonpregnant woman, which of the following parameters is most likely to be decreased in this patient?

- ☐ A. Cardiac preload
- ☐ B. Cardiac output
- ☐ C. Central venous pressure
- ☐ D. Plasma volume
- ☐ E. Systemic vascular resistance

Submit

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- A. Cardiac preload (11%)

✖

B. Cardiac output (3%)

C. Central venous pressure (9%)

D. Plasma volume (5%)

✔

E. Systemic vascular resistance (69%)
- Incorrect

Correct answer
E

69%

Answered correctly

03 secs

Time Spent

2023

Version

Explanation

Maternal cardiopulmonary adaptations to pregnancy	
	<ul style="list-style-type: none">• Cardiac<ul style="list-style-type: none">◦ ↑ Cardiac output◦ ↑ Plasma volume

Maternal cardiopulmonary adaptations to pregnancy	
Maternal adaptations	<ul style="list-style-type: none">• Cardiac<ul style="list-style-type: none">◦ ↑ Cardiac output◦ ↑ Plasma volume◦ ↓ Systemic vascular resistance• Respiratory<ul style="list-style-type: none">◦ ↑ Tidal volume◦ ↓ Functional residual capacity (elevation of diaphragm)
Clinical manifestations	<ul style="list-style-type: none">• Peripheral edema• ↓ Blood pressure• ↑ Heart rate• Systolic ejection murmur• Dyspnea

During **pregnancy**, the maternal cardiovascular system undergoes **physiologic adaptations** to increase cardiac output and optimize fetal perfusion. The most significant hemodynamic change is a **marked decrease** in **systemic vascular resistance** (SVR), which occurs due to increased release of peripheral vasodilators (eg, nitric oxide, prostacyclin) and decreased vascular sensitivity to vasoconstrictors (eg, norepinephrine, angiotensin II). The formation of a high-flow, low-resistance circuit within the uterus and placenta also contributes to decreased SVR.

Decreased SVR induces mild hypotension, which along with direct stimulation by placental hormones, leads to activation of the renin-angiotensin-aldosterone system. This results in fluid retention and an **increase in plasma blood volume** of up to 40% in pregnancy (**Choice D**). This physiologic increase in plasma blood volume inevitably results in increased venous return (ie, increased cardiac preload), which together with reduced SVR (ie, decreased

During **pregnancy**, the maternal cardiovascular system undergoes **physiologic adaptations** to increase cardiac output and optimize fetal perfusion. The most significant hemodynamic change is a **marked decrease** in **systemic vascular resistance** (SVR), which occurs due to increased release of peripheral vasodilators (eg, nitric oxide, prostacyclin) and decreased vascular sensitivity to vasoconstrictors (eg, norepinephrine, angiotensin II). The formation of a high-flow, low-resistance circuit within the uterus and placenta also contributes to decreased SVR.

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Pulmonary vascular resistance (PVR) also decreases during pregnancy, likely via the same vasodilatory mechanisms as SVR. This helps accommodate the increased preload to the right heart while maintaining a **normal central venous pressure** (CVP) (**Choice C**). Vascular capacitance (ie, the ability of systemic veins to stretch) also helps accommodate increased total blood volume without affecting the CVP.

Despite normal CVP, **peripheral edema** is common in pregnancy due to the increase in plasma volume resulting in **decreased blood oncotic pressure**; this change encourages leakage of intravascular fluid into dependent peripheral tissue (eg, lower extremities).

Educational objective:

During pregnancy, systemic vascular resistance decreases and plasma blood volume increases. Venous capacitance and a decrease in pulmonary vascular resistance allow for accommodation of increased blood volume without affecting central venous pressure (CVP). Despite a normal CVP, peripheral edema is common in pregnancy due to decreased capillary oncotic pressure that encourages leakage of fluid into dependent peripheral tissues (eg, lower extremities).

A 3-week-old girl is brought to the emergency department due to lethargy. The patient was breastfeeding well until this morning when she became increasingly difficult to rouse. She was born at term to a 35-year-old woman who had a spontaneous vaginal delivery at home. The patient did not receive any vaccinations or medications after birth due to parental preference. Head circumference is at the 99th percentile. Weight and length are at the 25th percentile. Temperature is 37 C (98.6 F). Physical examination shows a large, bulging anterior fontanelle. The eyes are driven downward, and the patient does not appear able to look upward. No scalp swelling is present. Intracranial hemorrhage is confirmed on CT scan of the head. Which of the following is the most likely underlying cause of this patient's condition?

- ☐ A. Germinal matrix fragility
- ☐ B. Inherited hemophilia
- ☐ C. Perinatally acquired infection
- ☐ D. Trauma during birth
- ☐ E. Vitamin deficiency

Submit

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- A. Germinal matrix fragility (21%)
- ✗

B. Inherited hemophilia (1%)
- C. Perinatally acquired infection (20%)
- D. Trauma during birth (7%)
- ✓

E. Vitamin deficiency (49%)

Incorrect

Correct answer
E

49%
Answered correctly

04 secs
Time Spent

2023
Version

Explanation

Infantile vitamin K–deficient bleeding	
Pathophysiology	<ul style="list-style-type: none">Low vitamin K stores (poor placental transfer, sterile gut, low content in breast milk)

Infantile vitamin K–deficient bleeding	
Pathophysiology	<ul style="list-style-type: none">Low vitamin K stores (poor placental transfer, sterile gut, low content in breast milk)Inefficient vitamin K use by immature liver
Clinical features	<ul style="list-style-type: none">Classically presents on day 2-7 of life*Easy bruisingUmbilical, mucosal & gastrointestinal bleedingIntracranial hemorrhage
Laboratory findings	<ul style="list-style-type: none">↑ PT↑ PTT (if severe)Normal platelet count
Prevention	<ul style="list-style-type: none">Intramuscular vitamin K at birth
*Can occur up to age 6 months.	

This neonate who did not receive any medications after birth has an **intracranial hemorrhage** (ICH), most likely due to **vitamin K deficiency**.

Vitamin K is an essential cofactor for gamma-glutamyl carboxylase, the enzyme responsible for carboxylation of clotting factors II (prothrombin), VII, IX, and X. Healthy children and adults typically have adequate vitamin K stores from dietary sources (eg, green, leafy vegetables) and from endogenous colonic bacteria that generate the vitamin. In contrast, **newborns** have low vitamin K stores due to **poor transplacental transfer** and **low content in breast milk**. Delayed vitamin K production by the gradual colonization of bacterial gut flora as well as inefficient use by an immature liver also contribute to low levels in early infancy.

Neonates typically receive prophylactic intramuscular vitamin K shortly after birth to prevent vitamin K deficiency.

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Neonates typically receive prophylactic intramuscular vitamin K shortly after birth to prevent vitamin K deficiency. Those who do not receive prophylaxis, like this patient, are at risk for **bleeding complications** (eg, umbilical/gastrointestinal bleeding) caused by **impaired clotting factor activity**. ICH is a potentially fatal complication and presents with signs of **increased intracranial pressure** (eg, lethargy, enlarging head circumference, bulging fontanel, downward-driven eyes). Vitamin K–deficient bleeding usually occurs in the first week of life but can present through age 6 months.

(Choice A) The germinal matrix is a highly vascularized periventricular area with poor structural integrity in utero. Its fragility is often responsible for **intraventricular hemorrhage** in preterm infants, but the region involutes around 28 weeks gestation, making it an unlikely cause of bleeding in a term infant.

(Choice B) Hemophilia (factor VIII or IX deficiency) can present with neonatal ICH but is an X-linked recessive disorder primarily affecting males. Females who are heterozygous can have mild symptoms due to skewed lyonization, but life-threatening bleeds do not occur.

(Choice C) Lethargy and a bulging fontanelle in a neonate raises concern for bacterial meningitis (eg, group B *Streptococcus*), but ICH would not be expected and temperature instability (eg, fever, hypothermia) is often seen. Other infections, such as congenital toxoplasmosis and cytomegalovirus, can cause intracranial calcifications, not ICH.

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(Choice D) ICH from birth injury is most common in operative deliveries (eg, vacuum/forceps assistance), not seen here. Neonates would be symptomatic shortly after birth rather than at age 3 weeks.

Educational objective:

Neonates are at risk for vitamin K deficiency primarily due to poor transplacental transfer and low content in breast milk. Because vitamin K is necessary for clotting factor activity, neonates who do not receive vitamin K prophylaxis at birth are predisposed to bleeding complications such as intracranial hemorrhage.

This neonate who did not receive any medications after birth has an **intracranial hemorrhage (ICH)**, most likely due to **vitamin K deficiency**.

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Exhibit Display

Signs of increased intracranial pressure

Wide, bulging fontanel

Prominent scalp veins

Increased head circumference

Sunset eyes

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Exhibit Display

Germinal matrix hemorrhage

Lateral ventricle

Germinal matrix

Hemorrhage extending into lateral ventricle

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A 28-year-old woman, gravida 2 para 1, at 18 weeks gestation comes to the office for a routine prenatal appointment. She is feeling well and has had no complications during this pregnancy. The patient has no chronic medical conditions and has had no previous surgeries. Her only medications are a daily prenatal vitamin and an iron supplement. Blood pressure is 110/70 mm Hg and pulse is 62/min. BMI is 24 kg/m². Cardiopulmonary examination is unremarkable. The uterus is consistent with 18 weeks gestation and fetal heart tones are normal. There is mild bilateral pedal edema to the shins. Compared to a nonpregnant state, which of the following changes are most likely expected in this patient?

	Fibrinogen level	Protein S level	Fibrinolysis activity
<input type="radio"/> A.	↓	↑	↑
<input type="radio"/> B.	↓	↑	↓
<input type="radio"/> C.	↑	No change	↑
<input type="radio"/> D.	↑	↓	↓
<input type="radio"/> E.	No change	↑	↓
<input type="radio"/> F.	No change	↓	↑

Submit

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<input type="radio"/> A.	↓	↑	↑	(13%)
<input checked="" type="radio"/> B.	↓	↑	↓	(7%)
<input type="radio"/> C.	↑	No change	↑	(11%)
<input checked="" type="radio"/> D.	↑	↓	↓	(54%)
<input type="radio"/> E.	No change	↑	↓	(9%)
<input type="radio"/> F.	No change	↓	↑	(3%)

Incorrect

Correct answer
D

54%

Answered correctly

04 secs

Time Spent

2023

Version

Explanation

Physiologic changes of pregnancy	
Cardiovascular	<ul style="list-style-type: none">• ↑ Blood volume (plasma > RBC mass)• ↓ Systemic vascular resistance• ↑ Heart rate & cardiac output
Pulmonary	<ul style="list-style-type: none">• ↑ Central respiratory drive (hyperventilation)• ↓ PaCO₂ (respiratory alkalosis), ↑ PaO₂
Renal	<ul style="list-style-type: none">• ↑ Renal blood flow & urine output• ↑ GFR, ↓ BUN & serum creatinine• ↑ HCO₃⁻ excretion (metabolic compensation)• ↓ Serum Na⁺ concentration (↑ ADH secretion)
Hematologic	<ul style="list-style-type: none">• ↑ Prothrombotic coagulation factors• ↓ Hemoglobin concentration (dilutional anemia)
ADH = antidiuretic hormone; BUN = blood urea nitrogen; GFR = glomerular filtration rate; RBC = red blood cell.	

Normal pregnancy is associated with physiologic changes to the maternal clotting system in order to adapt to pregnancy and prepare for delivery. In general, pregnancy is a mild **prothrombotic state** due to the following:

- **Increased procoagulant factors:** coagulation factors involved in the intrinsic (eg, factor XII), extrinsic (eg, factor VII), and final common (eg, **fibrinogen**) pathways of clotting are increased by up to 200%, which promotes the formation of cross-linked fibrin clots.
- Decreased anticoagulant factors: **protein S levels decrease** and resistance to activated protein C increases, leading to reduced clotting factor proteolysis.
- **Reduced fibrinolysis:** activity of fibrinolytic inhibitors (eg, plasminogen activator inhibitor-1 derived from the placenta) increases significantly, which reduces the breakdown of fibrin clots.

Renal	<ul style="list-style-type: none">• ↑ Renal blood flow & urine output• ↑ GFR, ↓ BUN & serum creatinine• ↑ HCO₃⁻ excretion (metabolic compensation)• ↓ Serum Na⁺ concentration (↑ ADH secretion)
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- Decreased anticoagulant factors: **protein S levels decrease** and resistance to activated protein C increases, leading to reduced clotting factor proteolysis.
- **Reduced fibrinolysis:** activity of fibrinolytic inhibitors (eg, plasminogen activator inhibitor-1 derived from the placenta) increases significantly, which reduces the breakdown of fibrin clots.

Although these changes protect against excessive maternal bleeding during fetal delivery and separation of the placenta, they also increase the risk of peripartum venous thromboembolism.

Educational objective:

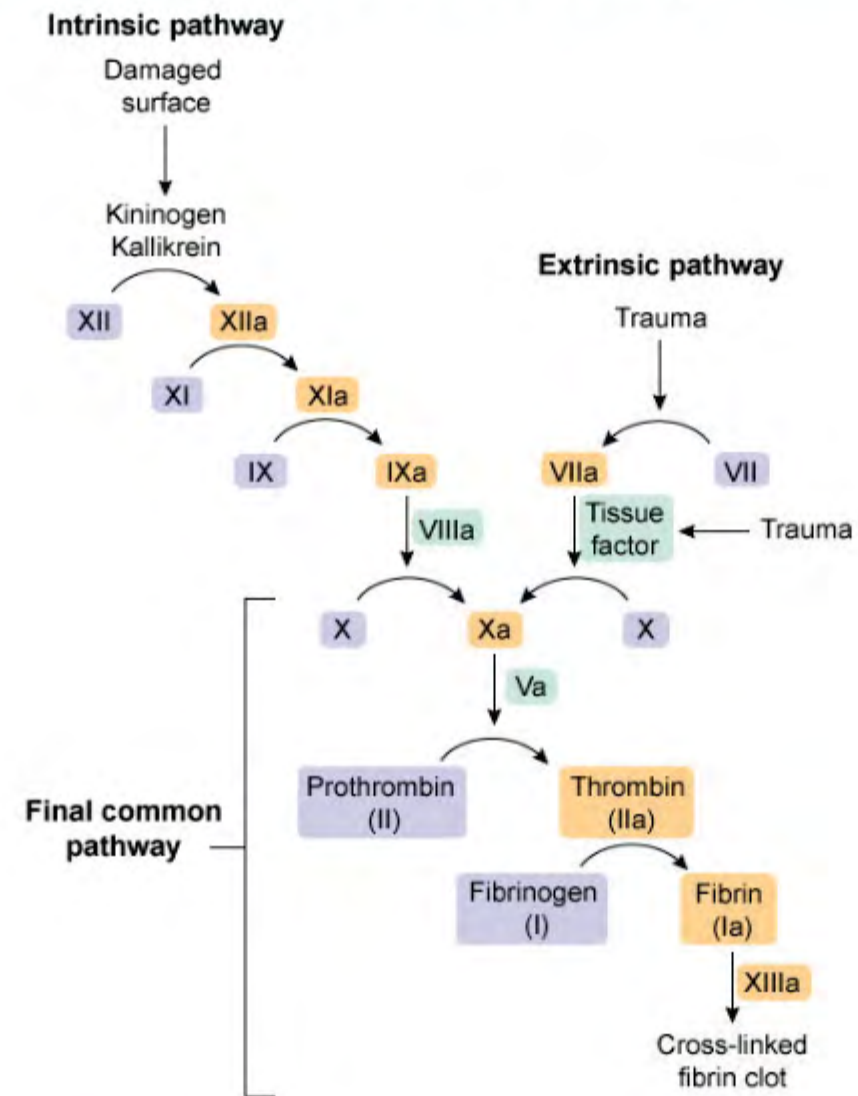
Pregnancy is a prothrombotic state due to increased procoagulant factors (eg, fibrinogen), decreased anticoagulant factors (eg, protein S), and reduced fibrinolysis. These physiologic changes protect against maternal hemorrhage with delivery but increase the risk of peripartum venous thromboembolism.

References

- ↓ Serum Na⁺ concentration (↑ ADH secretion)

Exhibit Display

Coagulation cascade pathway



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A 36-year-old woman, gravida 2 para 1, at 34 weeks gestation is brought to labor and delivery due to heavy vaginal bleeding. She has had no contractions, leakage of fluid, or recent trauma. The patient has received no prenatal care. Her first pregnancy ended in a full-term cesarean delivery. Temperature is 37.2 C (99 F), blood pressure is 134/86 mm Hg, and pulse is 98/min. On speculum examination, there is active heavy bleeding. An emergency cesarean delivery is performed. After delivery of the infant, the placenta cannot be removed and remains densely adherent to the uterine wall. An emergency hysterectomy is performed. Pathologic examination will most likely show which of the following?

- ☐ A. Abnormal placental trophoblast proliferation
- ☐ B. Absence of the endometrial decidua basalis
- ☐ C. Absence of Wharton jelly at cord insertion
- ☐ D. Infiltration of neutrophils in the umbilical cord

Submit

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- ☐ A. Abnormal placental trophoblast proliferation (52%)

☒ B. Absence of the endometrial decidua basalis (41%)

☐ C. Absence of Wharton jelly at cord insertion (4%)

☐ D. Infiltration of neutrophils in the umbilical cord (1%)

Correct

41%

Answered correctly

04 secs

Time Spent

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Explanation



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This patient with a placenta adherent to the uterine wall likely had **placenta accreta**, the abnormal attachment of placental villi directly to the uterine myometrium rather than to the endometrial decidua basalis.

The uterus normally consists of 3 distinct layers: serosa, myometrium, and endometrium. The endometrium is subdivided into the stratum functionalis and stratum basalis. In early pregnancy, progesterone stimulates decidualization of the stratum basalis, forming the endometrial decidua basalis.

Normally, fetal trophoblasts that form the placental villi attach to the endometrial decidua basalis. However, patients with prior uterine surgery (eg, **cesarean delivery**) can have **endometrial scarring** or defects that impair normal decidualization. In these patients, the **absent decidua basalis** results in direct placental attachment to the myometrium and a morbidly adherent placenta.

Placenta accreta is usually diagnosed on routine prenatal ultrasonography; however, in some cases (eg, no prenatal care), the diagnosis is made after fetal delivery when there is difficulty detaching the placenta. Attempts to remove the placenta typically lead to **postpartum hemorrhage** due to the disruption of highly vascular adhesions between the uterus and placenta.

(Choice A) Abnormal placental trophoblast proliferation is seen in gestational trophoblastic disease (eg, complete hydatidiform mole), which typically presents with vaginal bleeding and uterine enlargement. It does not cause a densely adherent placenta.

(Choice C) Wharton jelly is the gelatinous tissue surrounding the umbilical vessels that protects them from injury. Absence of Wharton jelly at the cord insertion site may be seen with **vasa previa**, in which the umbilical vessels become vulnerable to injury.

(Choice D) Infiltration of neutrophils into the umbilical cord may be seen with intraamniotic infection. However, additional signs of infection (eg, fever, purulent amniotic fluid) would likely be present and the placenta would not be densely adherent.

Educational objective:

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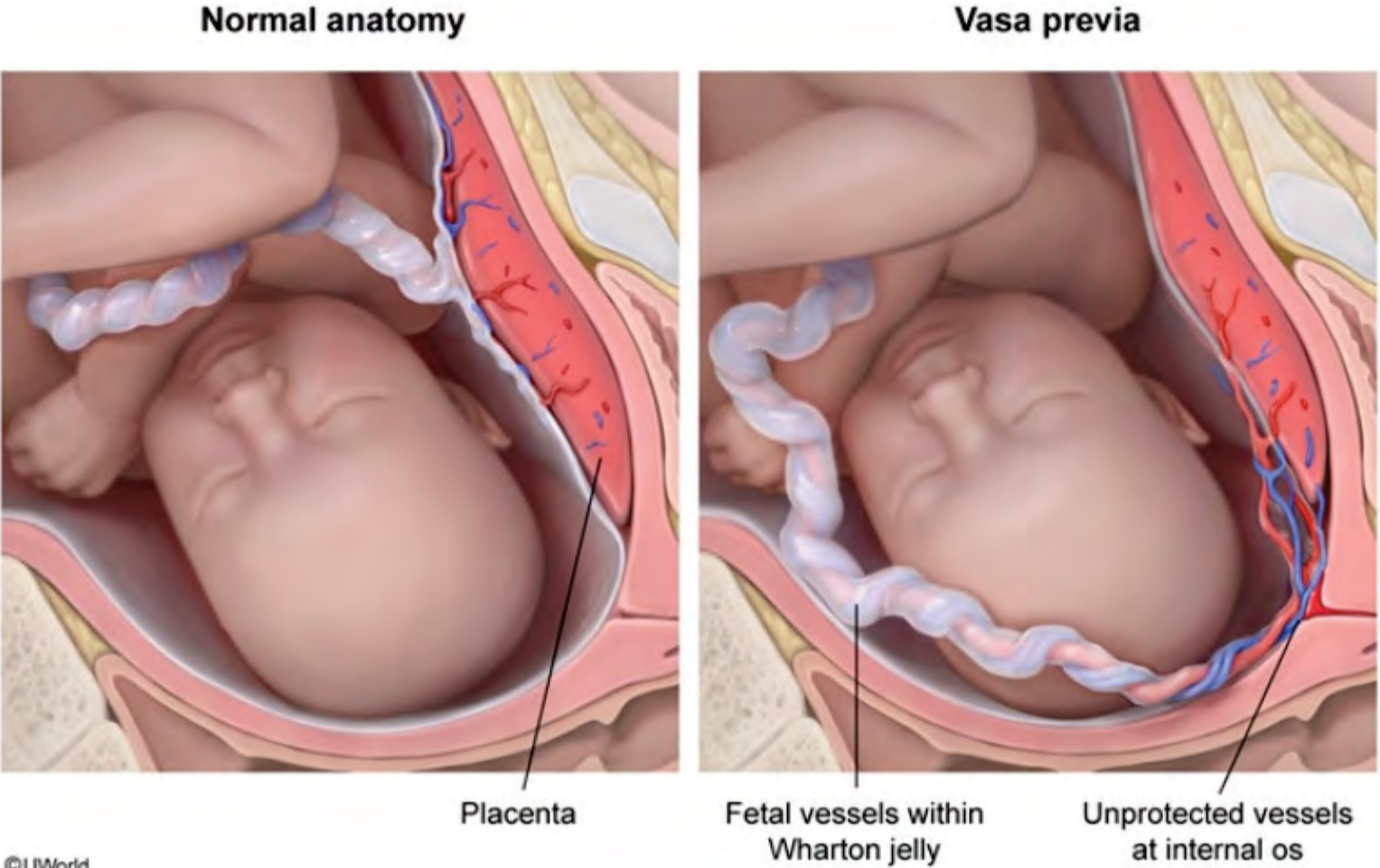
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Educational objective:

Placenta accreta occurs due to abnormal attachment of the placenta directly onto the uterine myometrium. This condition most often develops because the endometrial decidua basalis is absent or defective due to previous uterine scarring (eg, prior cesarean delivery). The classic presentation is a morbidly adherent placenta that does not detach after fetal delivery.

placental villi directly to the uterine myometrium rather than to the endometrial decidua basalis.

The Exhibit Display



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Placenta accreta occurs due to abnormal attachment of the placenta directly onto the uterine myometrium. This

A 35-year-old woman at 38 weeks gestation undergoes induction of labor. The patient has type 2 diabetes mellitus, and her blood glucose levels were increasingly difficult to control during her third trimester despite increasing doses of insulin. During induction, the patient develops arrest of labor and undergoes a cesarean delivery. Apgar scores are 8 and 9 at 1 and 5 minutes, respectively. Physical examination shows a newborn large for gestational age with a birth weight of 4.6 kg (10.1 lb). The remainder of the examination is unremarkable, and vital signs are normal. An hour after delivery, the newborn develops hypoglycemia. Which of the following is most likely the cause of this newborn's hypoglycemia?

- ☐ A. Excessive fetal insulin production
- ☐ B. Excessive maternal insulin doses
- ☐ C. Fetal cortisol deficiency
- ☐ D. Impaired fetal glucose production
- ☐ E. Impaired fetal insulin-like growth factor 2 production
- ☐ F. Inadequate fetal hepatic glycogen stores

Submit

A 35-year-old woman at 38 weeks gestation undergoes induction of labor. The patient has type 2 diabetes mellitus, and her blood glucose levels were increasingly difficult to control during her third trimester despite increasing doses of insulin. During induction, the patient develops arrest of labor and undergoes a cesarean delivery. Apgar scores are 8 and 9 at 1 and 5 minutes, respectively. Physical examination shows a newborn large for gestational age with a birth weight of 4.6 kg (10.1 lb). The remainder of the examination is unremarkable, and vital signs are normal. An hour after delivery, the newborn develops hypoglycemia. Which of the following is most likely the cause of this newborn's hypoglycemia?

- ✓

☐

A. Excessive fetal insulin production (76%)
- ✗

☒

B. Excessive maternal insulin doses (13%)
- ☐

C. Fetal cortisol deficiency (1%)
- ☐

D. Impaired fetal glucose production (4%)
- ☐

E. Impaired fetal insulin-like growth factor 2 production (1%)
- ☐

F. Inadequate fetal hepatic glycogen stores (2%)

Incorrect

Correct answer
A

76%

Answered correctly

03 secs

Time Spent

2023

Version

Explanation

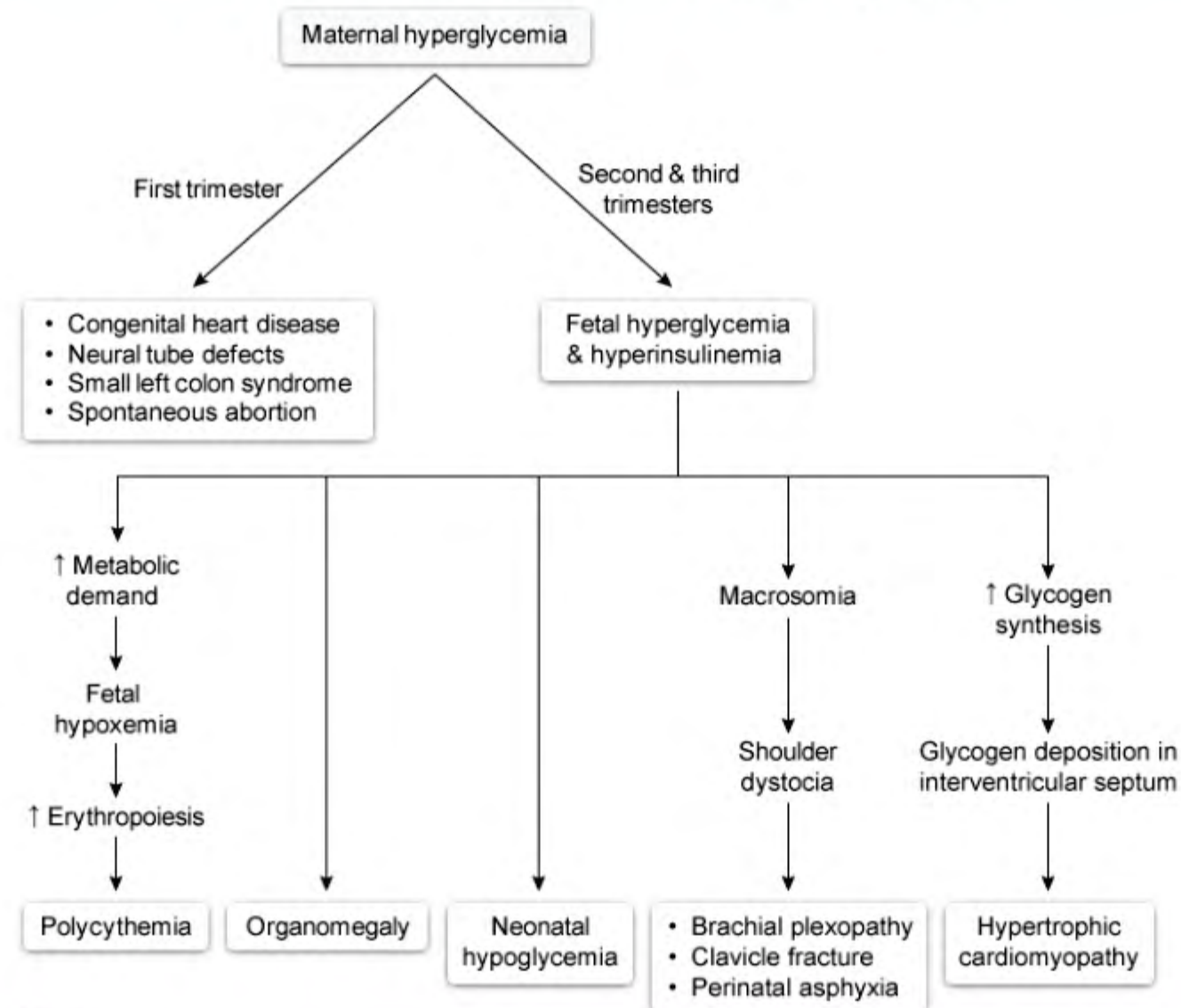
Infant of mother with diabetes mellitus: complications

Maternal hyperglycemia

☐ D. Impaired fetal glucose production (4%)

Exhibit Display

Infant of mother with diabetes mellitus: complications



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Diabetes mellitus in pregnancy is associated with a number of neonatal complications. In pregnant women with poorly controlled diabetes mellitus, the fetus is subjected to high blood glucose levels because glucose freely crosses the placenta. The resulting **fetal hyperglycemia** leads to a compensatory **pancreatic beta cell hyperplasia** and subsequent increase in fetal insulin production (eg, hyperinsulinemia).

The resultant **fetal hyperinsulinemia** causes the following neonatal complications:

- **Hypoglycemia:** After delivery, the fetus is no longer exposed to high glucose levels; however, due to beta cell hyperplasia, the neonate continues to secrete high levels of insulin, resulting in symptomatic low glucose levels (eg, jitteriness, irritability)
- **Polycythemia:** Increased fetal metabolism and increased fetal oxygen demand, resulting in hypoxemia and a compensatory rise in fetal erythropoietin (ie, polycythemia)
- **Macrosomia:** Increased central adipose deposition (abdominal and interscapular), leading to increased delivery complications (eg, shoulder dystocia, cesarean delivery)

(Choice B) Maternal insulin is not transferred to the fetus but instead is catabolized by the placenta; therefore, even with increasing maternal insulin use, the neonate is not affected because of placental metabolism.

(Choice C) Cortisol deficiency in the newborn period is likely due to primary adrenal insufficiency, which would present with hypotension and electrolyte abnormalities in addition to hypoglycemia.

(Choice D) Glycogen storage diseases are caused by enzymatic defects that impair glycogenolysis and typically present with hypoglycemia and lactic acidosis. However, they are not evident immediately after birth because the neonates have frequent glucose resupply (from frequent breastfeeding) and, therefore, do not depend on the breakdown of glycogen.

(Choice E) Fetal hyperglycemia can result in increased secretion (not impaired production) of insulin-like growth factor 2 (IGF-2), causing macrosomia.

levels (eg, jitteriness, irritability)

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(Choice E) Fetal hyperglycemia can result in increased secretion (not impaired production) of insulin-like growth factor 2 (IGF-2), causing macrosomia.

(Choice F) Insulin promotes glucose storage in the liver as glycogen; therefore, infants of mothers with poorly controlled diabetes have increased glycogen stores. However, despite hypoglycemia, these infants have poor mobilization of glycogen (glycogenolysis) due to islet hyperplasia and the resultant hyperinsulinemia.

Educational objective:

Neonates born to mothers with poorly controlled diabetes during pregnancy are exposed to high maternal glucose levels in utero, resulting in beta cell hyperplasia followed by excessive fetal insulin production. Fetal hyperinsulinemia causes neonatal hypoglycemia, polycythemia, and macrosomia.

A 32-year-old woman, gravida 1 para 0, comes to the office to discuss abnormal laboratory results. The patient is at 18 weeks gestation based on her last menstrual period. She has no chronic medical conditions and takes no daily medications. Results of the quadruple screen performed previously are as follows:

Maternal serum α -fetoprotein	Low
Unconjugated estriol	Low
β -hCG	High
Inhibin A	High

Which of the following is the most likely diagnosis?

- ☐ A. Amniotic band sequence

☐ B. Gastroschisis

☐ C. Multiple gestation

☐ D. Myelomeningocele

☐ E. Trisomy 21

Submit

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Maternal serum α -fetoprotein	Low
Unconjugated estriol	Low
β -hCG	High
Inhibin A	High

Which of the following is the most likely diagnosis?

- A. Amniotic band sequence (5%)

✖

B. Gastroschisis (2%)

C. Multiple gestation (11%)

D. Myelomeningocele (5%)

✔

E. Trisomy 21 (74%)

Incorrect

Correct answer
E

74%
Answered correctly

06 secs
Time Spent

2023
Version

Explanation

Maternal serum α -fetoprotein screening	
\uparrow MSAFP	\downarrow MSAFP
<ul style="list-style-type: none">Open neural tube defects (eg, anencephaly, open spina bifida)Ventral wall defects (eg, omphalocele, gastroschisis)Multiple gestation	<ul style="list-style-type: none">Aneuploidies (eg, trisomy 18 & 21)
MSAFP = maternal serum α -fetoprotein.	

Trisomy 21, the cause of Down syndrome (DS), is the most common chromosomal abnormality and most common genetic cause of **intellectual disability**. Risk factors include **advanced maternal age (≥ 35)** and a parent with a Robertsonian translocation.

DS can be screened and diagnosed prenatally. One common option for screening during the second trimester is the **quadruple screen** at 15-19 weeks gestation. **Low maternal serum alpha-fetoprotein (AFP) and unconjugated estriol** levels are associated with DS and correlate with decreased fetal levels (possibly due to suboptimal fetal tissue function). In addition, **increased β -hCG and inhibin A** are secreted by the placenta, possibly due to compensatory placental hyperfunction. The diagnosis is confirmed by karyotyping fetal cells in the amniotic fluid (amniocentesis).

Most patients with DS have trisomy 21 from **meiotic nondisjunction**—specifically, failure of chromosome 21 to divide during meiosis. Chromosome 21 nondisjunction occurs during **oogenesis** and is significantly associated with advanced maternal age; it is not seen in spermatogenesis or in postzygotic mitotic errors. DS can also result from an **unbalanced Robertsonian translocation**, during which the entire long arm of one chromosome 21 is translocated to the long arm of an acrocentric chromosome (ie, chromosome 14). These individuals have 46 chromosomes; however, one chromosome 14 is comprised of the long arms of both chromosomes 14 and 21.

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(Choices A, B, and D) AFP is produced in both the yolk sac and developing fetal liver; fetal defects that disrupt the abdominal wall or neural tube allow AFP to concentrate in the amniotic fluid, thereby raising maternal serum levels. **Gastroschisis**, an abdominal wall defect lateral to the umbilicus with herniation of uncovered bowel, and **myelomeningocele**, a type of neural tube defect, are two conditions associated with an elevated maternal AFP. Amniotic bands, loose strands of amnion that constrict and disrupt developing fetal structures, can affect the abdominal wall or neural tube development in severe cases and lead to elevated AFP levels.

(Choice C) Multiple gestation will cause all of the quadruple screen analytes to be abnormally high due to increased fetal tissue and placental mass associated with ≥ 2 fetuses.

Educational objective:

DS can be screened and diagnosed prenatally. One common option for screening during the second trimester is the **quadruple screen** at 15-19 weeks gestation. **Low maternal serum alpha-fetoprotein (AFP) and unconjugated estriol** levels are associated with DS and correlate with decreased fetal levels (possibly due to suboptimal fetal tissue function). In addition, **increased β -hCG and inhibin A** are secreted by the placenta, possibly due to compensatory placental hyperfunction. The diagnosis is confirmed by karyotyping fetal cells in the amniotic fluid (amniocentesis).

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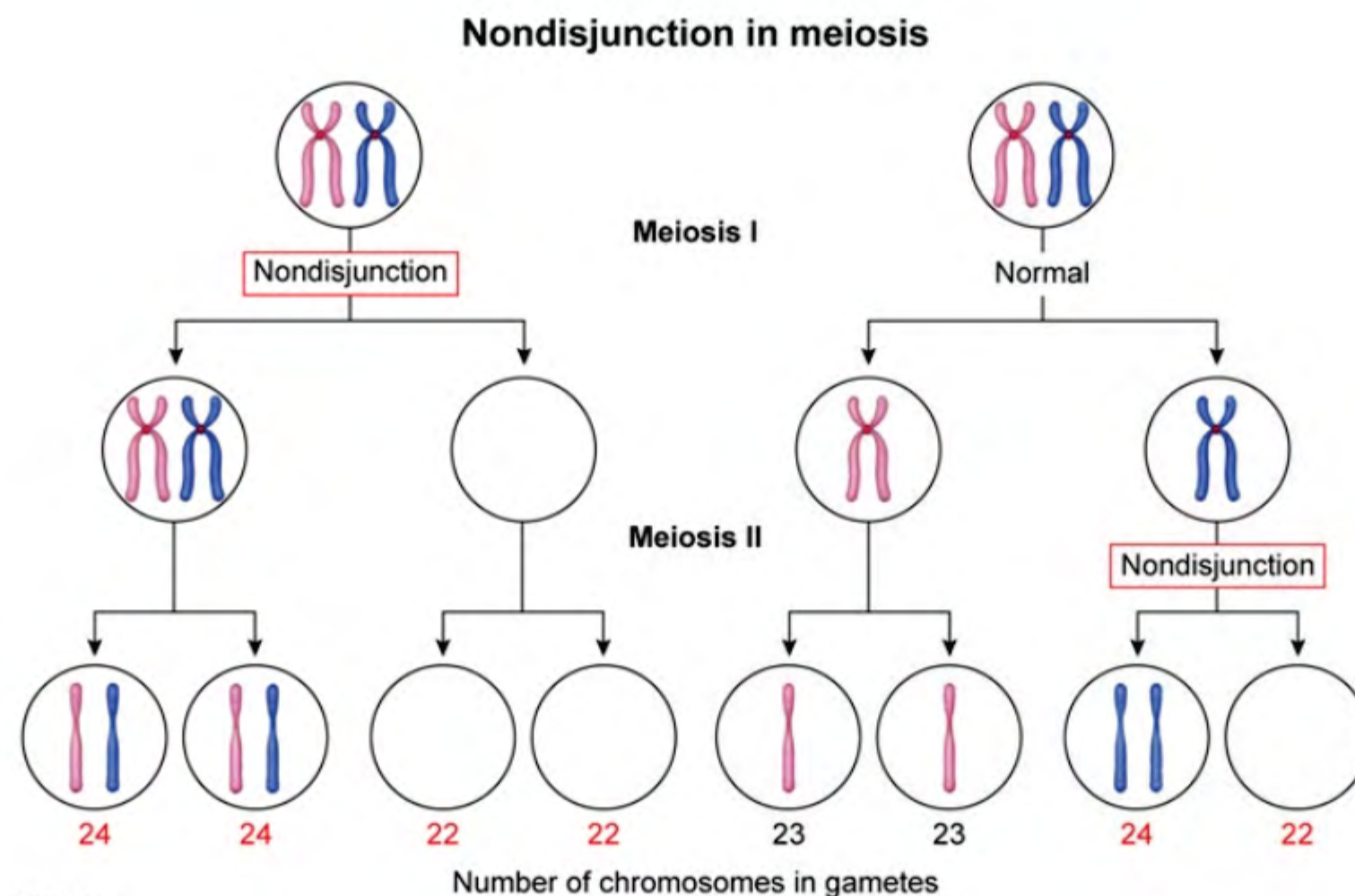
(Choice C) Multiple gestation will cause all of the quadruple screen analytes to be abnormally high due to increased fetal tissue and placental mass associated with ≥ 2 fetuses.

Educational objective:

Down syndrome is the most common chromosomal anomaly. It is associated with low levels of maternal serum alpha-fetoprotein (AFP) and estriol, while β -hCG and inhibin A levels are increased. Elevated AFP levels are seen in multiple gestation, open neural tube defects, and abdominal wall defects.

• Multiple gestation

Exhibit Display



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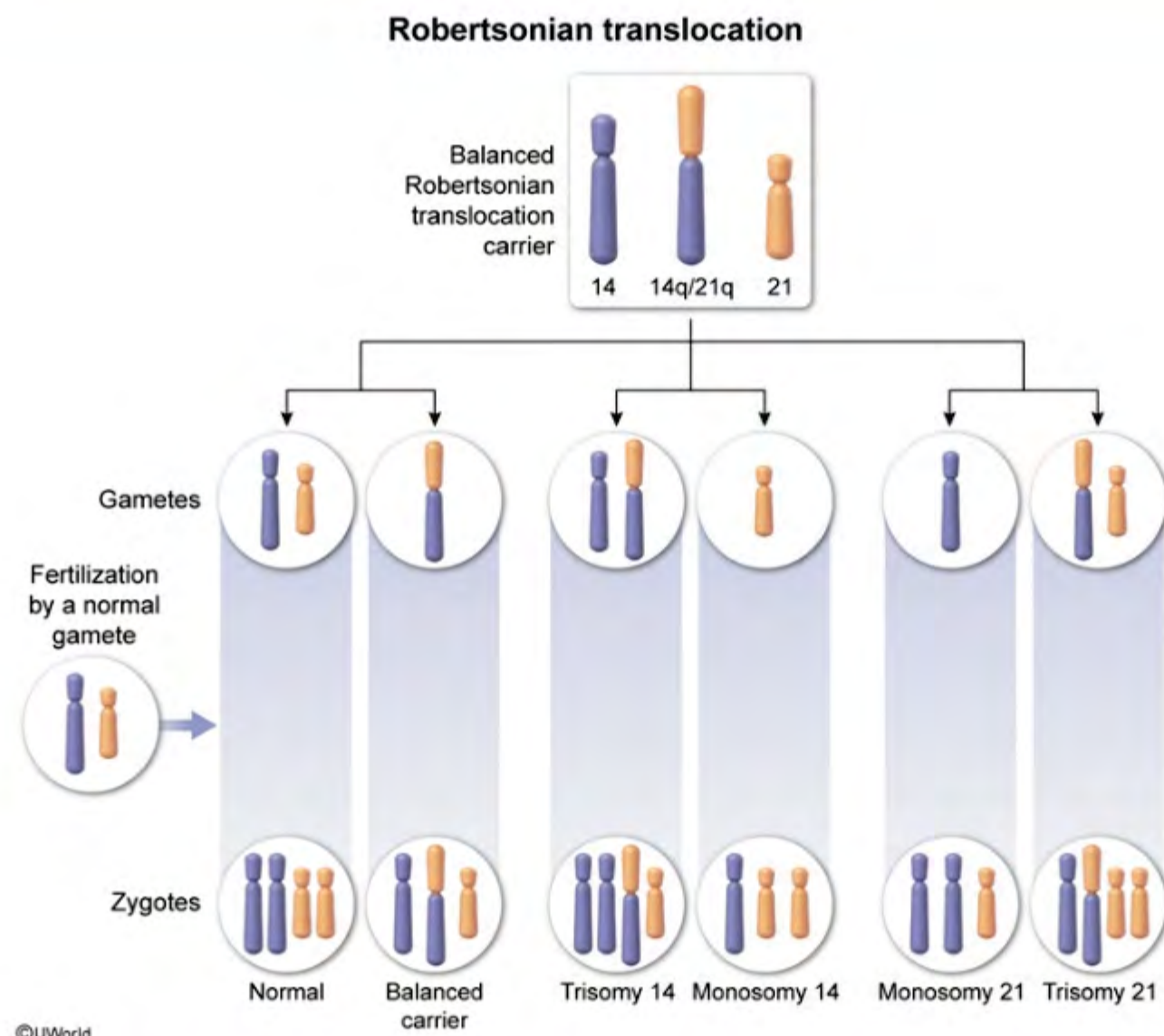
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<https://t.me/USMLEWorldStep1>

• Multiple gestation

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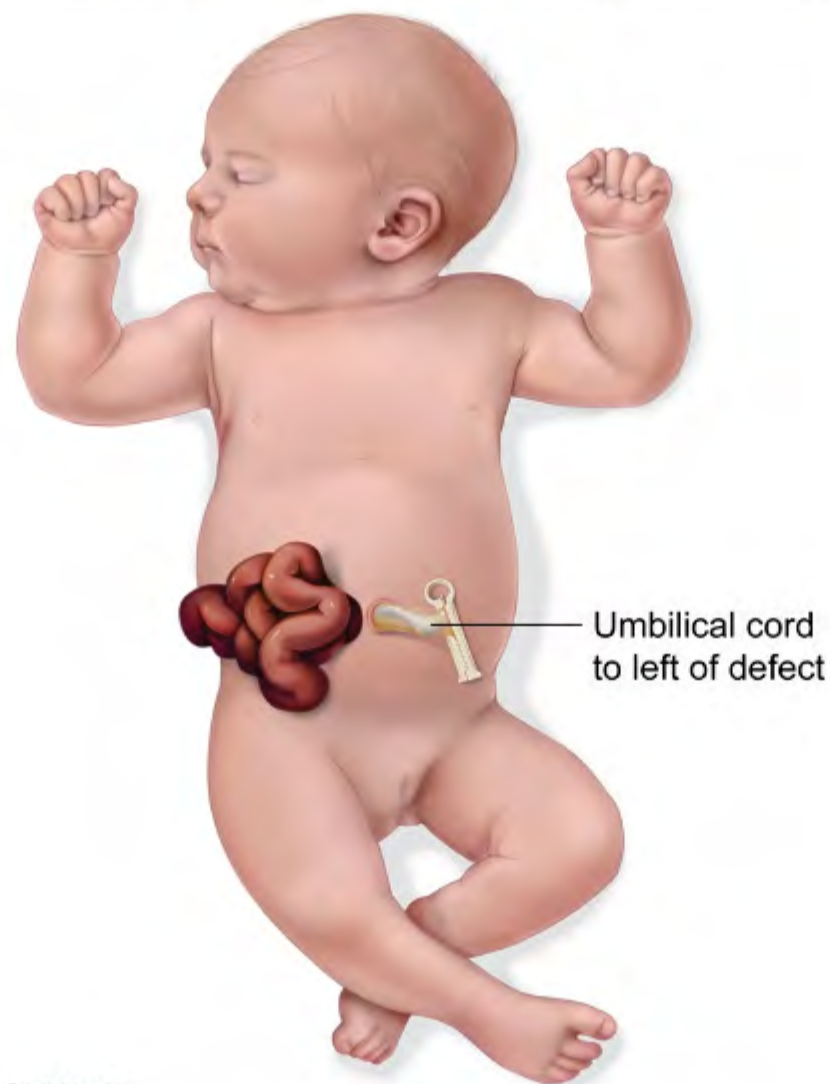
(Choice C) Multiple gestation will cause all of the quadruple screen analytes to be abnormally high due to

- Multiple gestation

Exhibit Display

Gastroschisis

Eviscerated bowel with no covering membrane



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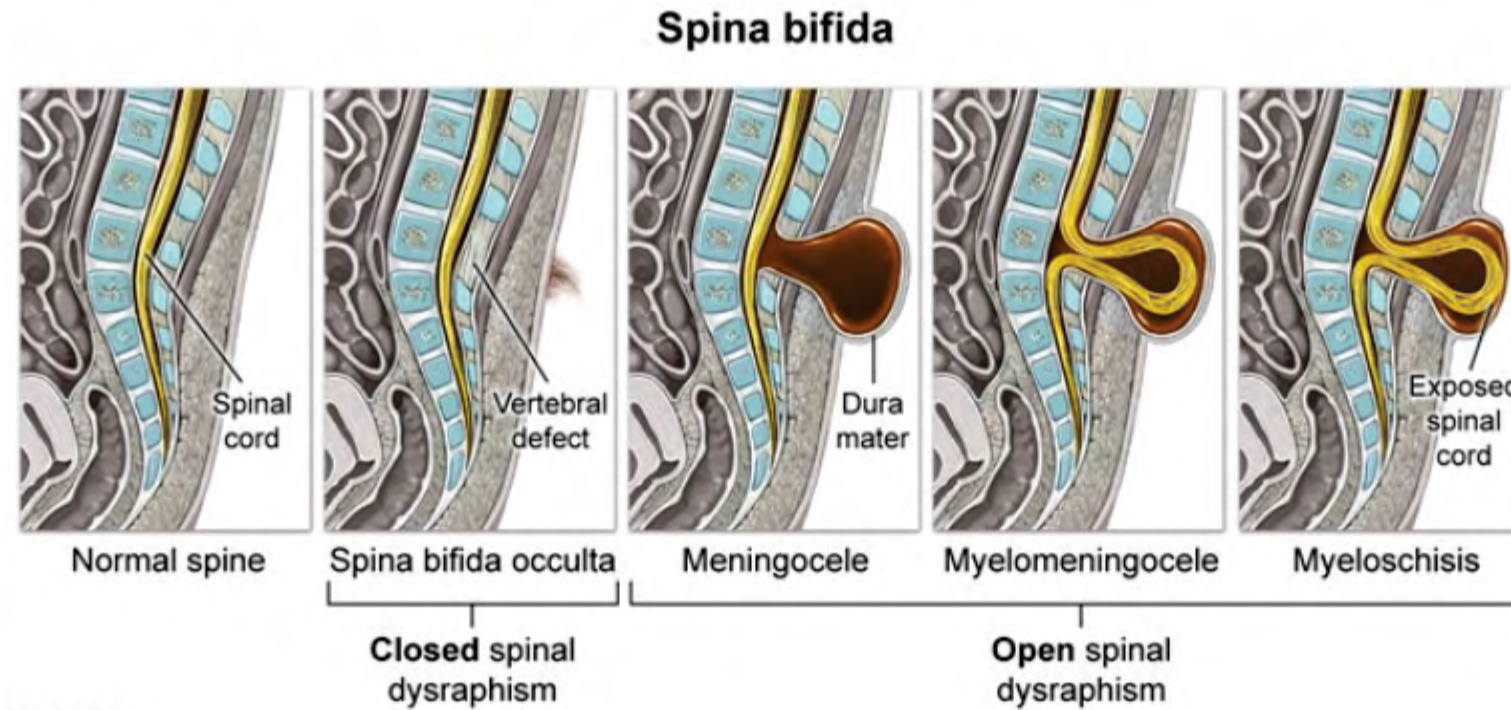
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<https://t.me/USMLEWorldStep1>



• Multiple gestation

Exhibit Display



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<https://t.me/USMLEWorldStep1>

A 33-year-old woman, gravida 0, comes to the office for preconception counseling. Menarche was at age 12, and her menses recur every 30 days and last 5 days. She has no chronic medical conditions, takes no medications, and has no allergies. BMI is 23 kg/m². Vital signs and physical examination are normal. If a normal pregnancy develops, which of the following processes most immediately precedes secretion of β -hCG into the maternal circulation?

- ☐ A. Cytotrophoblast development
- ☐ B. Morula development
- ☐ C. Oocyte fertilization
- ☐ D. Syncytiotrophoblast invasion
- ☐ E. Zygote formation

Submit

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- A. Cytotrophoblast development (15%)

✗

B. Morula development (6%)

C. Oocyte fertilization (7%)

D. Syncytiotrophoblast invasion (66%)

E. Zygote formation (4%)
- Incorrect

Correct answer
D

66%

Answered correctly

03 secs

Time Spent

2023

Version
- Explanation
- The diagram illustrates the process of blastocyst implantation in three stages.
1. **Blastocyst**: A spherical structure with an outer layer of cells called the **Trophoblast (trophectoderm)** and an inner cluster of cells called the **Embryoblast**. The space between them is the **Blastocoel (blastocyst cavity)**.
2. **Implantation 6-7 days after fertilization**: The blastocyst is shown attaching to the uterine wall. The **Cytotrophoblast** is shown secreting **hCG-H***.
3. **Developing villi**: The embryo is shown embedded in the uterine wall, with the **Amniotic cavity** and **Yolk sac** visible. The **Developing villi** are shown extending into the uterine wall.
- Block Time Elapsed: 00:09:42

My Notebook

Flashcards

Feedback

Suspend

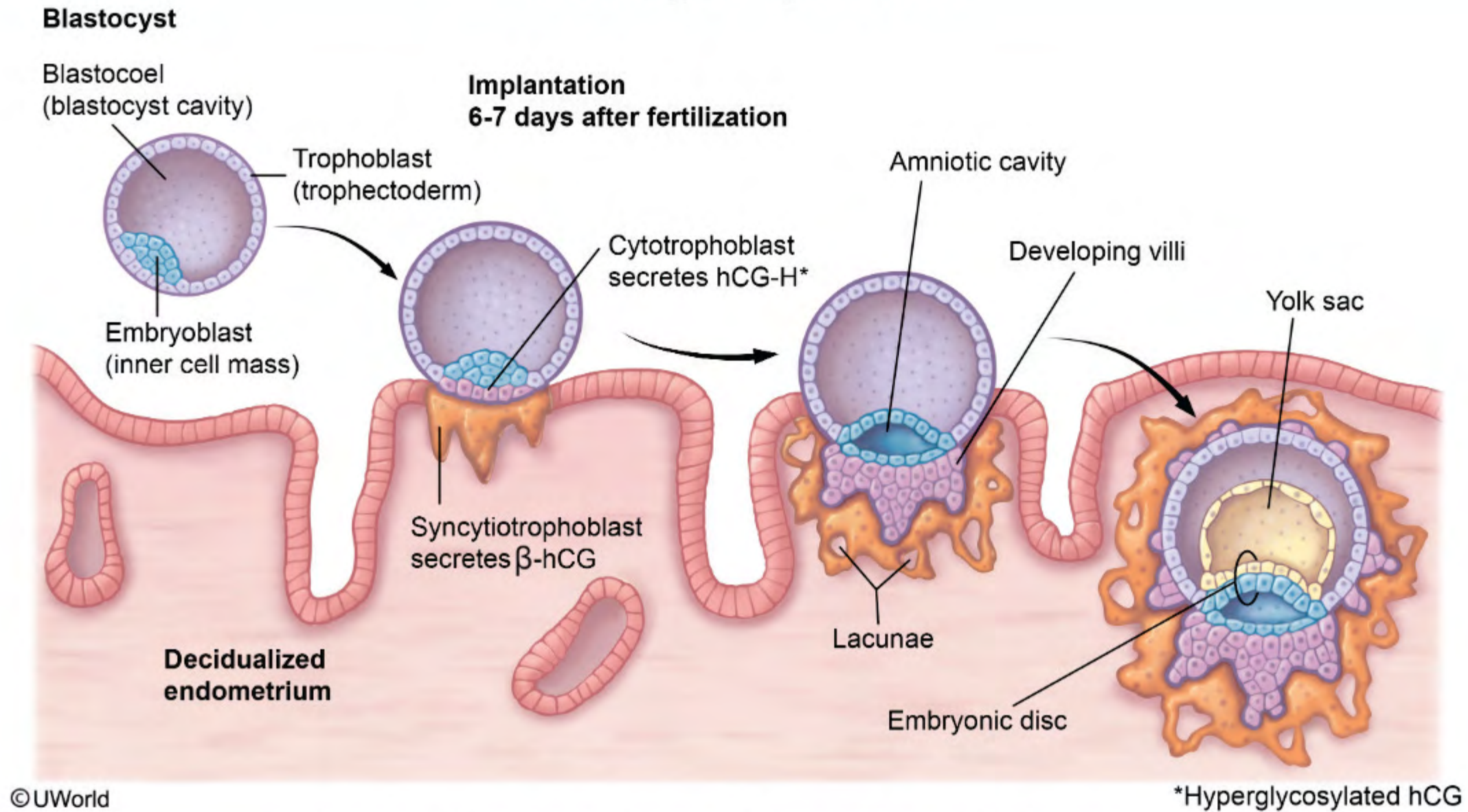
End Block

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A. Cytotrophoblast development (15%)

Exhibit Display

Blastocyst implantation



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β-hCG is a hormone that supports and **maintains early pregnancy** development. Levels begin to rise in the maternal serum approximately 8 days following fertilization and become detectable in the urine approximately 14 days following fertilization.

Oocyte fertilization within the ampulla of the fallopian tube initiates completion of the second meiotic division and zygote formation **(Choices C and E)**. The zygote then travels through the fallopian tube undergoing multiple mitotic divisions (cleavage), creating smaller cells (blastomeres) known collectively as a morula (a 16-32 cell mass) **(Choice B)**. About 3-4 days following fertilization, the morula enters the uterine cavity and also forms its own central cavity, converting it into a **blastocyst**.

Cellular differentiation within the blastocyst forms an inner cell mass (embryoblast) and an **outer layer of trophoblasts** (trophectoderm). The trophectoderm contains 2 different cell populations: undifferentiated cytotrophoblasts (ie, placental stem cells) and differentiated **syncytiotrophoblasts** that **invade the endometrium**. Following invasion, the syncytiotrophoblasts **begin secreting β-hCG** and other hormones (eg, progesterone, human placental lactogen) into the maternal circulation.

β-hCG prevents corpus luteum degradation, thereby sustaining ovarian progesterone production to help maintain the endometrium in a secretory state that supports early pregnancy. After 10 weeks of gestation, the placental mass and syncytiotrophoblasts volume have grown large enough to become the dominant source of progesterone production.

(Choice A) Cytotrophoblasts secrete hyperglycosylated hCG, a form of hCG distinct in function from β-hCG that promotes villous invasion in early pregnancy.

Educational objective:

β-hCG secretion begins with blastocyst implantation and syncytiotrophoblast invasion. Syncytiotrophoblast cells arise from the outer layer of the blastocyst (trophectoderm) and produce β-hCG, which maintains corpus luteum progesterone production and supports the developing early pregnancy.

An infant born to a 26-year-old woman is evaluated shortly after delivery. Birth weight and length are at the 10th and 15th percentiles, respectively. Vital signs are normal. Physical examination shows a protruding tongue, excessive skin at the nape of the neck, and upslanting palpebral fissures. The startle reflex is symmetric and weak. Cardiac auscultation reveals a harsh, III/VI systolic murmur heard best over the lower left sternal border. The patient has normal external female genitalia. Review of maternal medical records shows a past history of 2 spontaneous abortions in the last 3 years. Echocardiography confirms the presence of a ventricular septal defect. Which of the following karyotypes is most likely to be found in this infant?

- ☐ A. 45,XO
- ☐ B. 46,XX, del(22)(q11)
- ☐ C. 46,XX, t(9;22)
- ☐ D. 46,XX, t(14;21)
- ☐ E. 47,XX, +18

Submit

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- ✗

☒ A. 45,XO (29%)
- ☐ B. 46,XX, del(22)(q11) (7%)
- ☐ C. 46,XX, t(9;22) (1%)
- ✓

☐ D. 46,XX, t(14;21) (45%)
- ☐ E. 47,XX, +18 (15%)

Incorrect

Correct answer D

45% Answered correctly

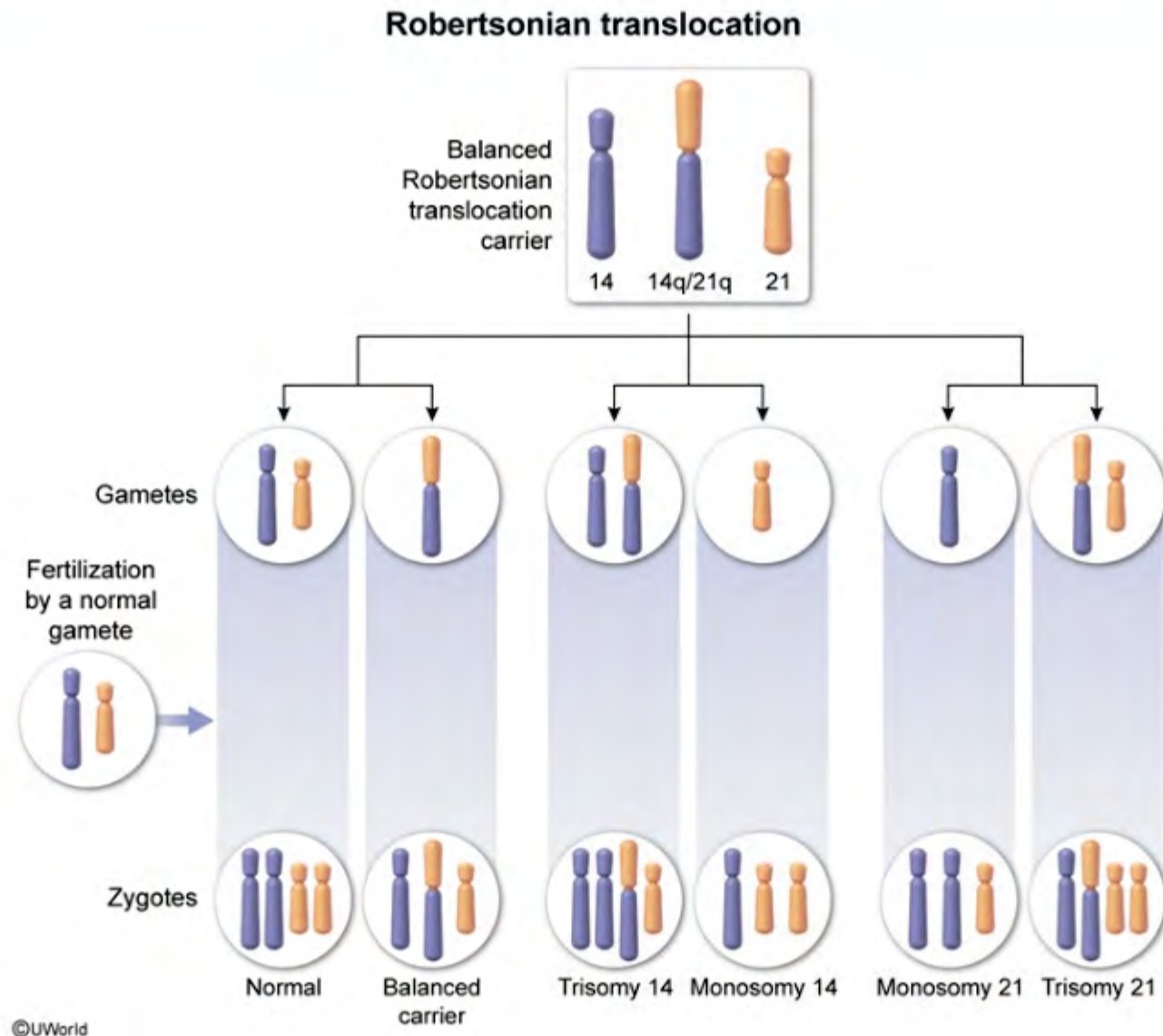
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2023 Version

Explanation



Exhibit Display



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Down syndrome is most commonly caused by random **meiotic nondisjunction**, an abnormality that correlates with increasing maternal age and results in **3 complete copies** of chromosome 21 (47, XX, +21).

Translocation Down syndrome, which is less common, can be inherited from an unaffected parent with a **balanced translocation**. Robertsonian translocations occur between 2 acrocentric, nonhomologous chromosomes (eg, 14 and 21). The resultant translocated chromosomes are the **fusion of 2 long arms** (eg, 14q and 21q [red arrow]) and the fusion of 2 short arms (eg, 14p and 21p); the latter usually contains nonessential genetic material and is lost after several cell divisions. The affected parent is asymptomatic because they have a normal (albeit rearranged) genetic complement. However, when an ovum containing the translocated chromosome and a normal chromosome 21 is fertilized with a sperm containing a normal set of chromosomes (ie, 1 copy of chromosome 21), the resultant fetus has an **unbalanced Robertsonian translocation** with **46 chromosomes** and **3 effective copies** of chromosome 21 [**46,XX, t(14;21)**]. Another possible maternal gamete includes 0 copies of chromosome 21, which increases the rate of **miscarriages** in balanced Robertsonian translocation carriers.

Dysmorphic features of Down syndrome include epicanthal folds, upslanting palpebral fissures, a protruding tongue, and excessive skin at the nape of the neck. Birth weight and length are often below average, and hypotonia and a weak startle (Moro) reflex are characteristic. Cardiac defects are present in >50% of cases.

(Choice A) **Turner syndrome** (45,XO) presents with low-set ears, a webbed neck, wide-spaced nipples, and cardiac defects (eg, aortic coarctation). Most cases are due to absence of the paternally contributed X chromosome.

(Choice B) DiGeorge syndrome, caused by sporadic 22q11 deletions, presents with hypertelorism, micrognathia, cleft palate, and cardiac defects (eg, interrupted aortic arch, Tetralogy of Fallot).

(Choice C) The Philadelphia chromosome is a balanced translocation between chromosomes 9 and 22 that produces an oncogenic fusion gene (*BCR-ABL*) associated with chronic myelogenous leukemia.

(Choice E) Trisomy 18 (47, XX, +18), or Edwards syndrome, usually arises secondary to meiotic nondisjunction

arrow]) and the fusion of 2 short arms (eg, 14p and 21p); the latter usually contains nonessential genetic material and is lost after several cell divisions. The affected parent is asymptomatic because they have a normal (albeit rearranged) genetic complement. However, when an ovum containing the translocated chromosome and a normal chromosome 21 is fertilized with a sperm containing a normal set of chromosomes (ie, 1 copy of chromosome 21), the resultant fetus has an **unbalanced Robertsonian translocation** with **46 chromosomes** and **3 effective copies** of chromosome 21 [**46,XX, t(14;21)**]. Another possible maternal gamete includes 0 copies of chromosome 21, which increases the rate of **miscarriages** in balanced Robertsonian translocation carriers.

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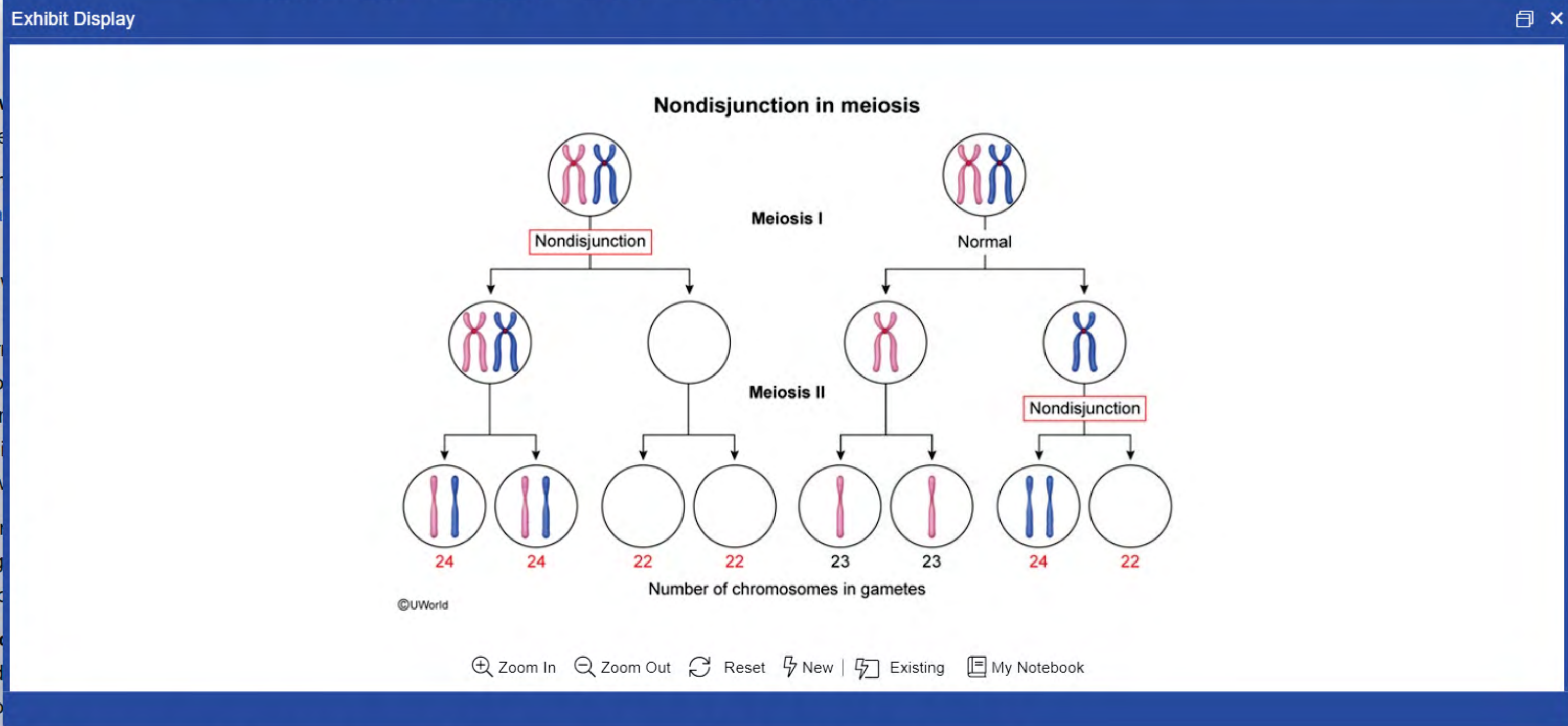
(Choice E) Trisomy 18 (47, XX, +18), or Edwards syndrome, usually arises secondary to meiotic nondisjunction with maternal age ≥35. Findings include dysmorphic facies (eg, micrognathia, low-set ears), clenched hands with overlapping fingers, and hypertonia.

Educational objective:

Unbalanced Robertsonian translocations account for a minority of Down syndrome cases. Karyotyping shows 46 chromosomes with a translocation between 2 acrocentric nonhomologous chromosomes [eg, 46, XX, t(14;21)].

Reference

Zygotes



(Choice B) DiGeorge syndrome, caused by sporadic 22q11 deletions, presents with hypertelorism, micrognathia, cleft palate, and cardiac defects (eg, interrupted aortic arch, Tetralogy of Fallot).

Zygotes

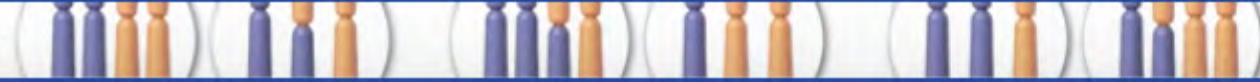
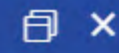


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Trisomy 21 (Down syndrome)



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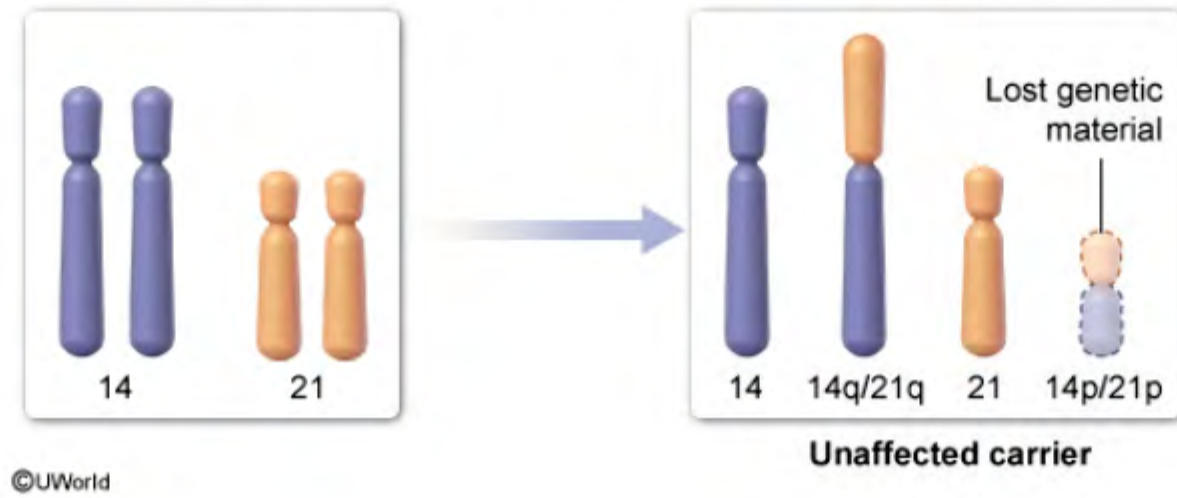
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Zygotes

Exhibit Display

Balanced Robertsonian translocation



Zoom In Zoom Out Reset New Existing My Notebook

Zygotes



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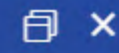
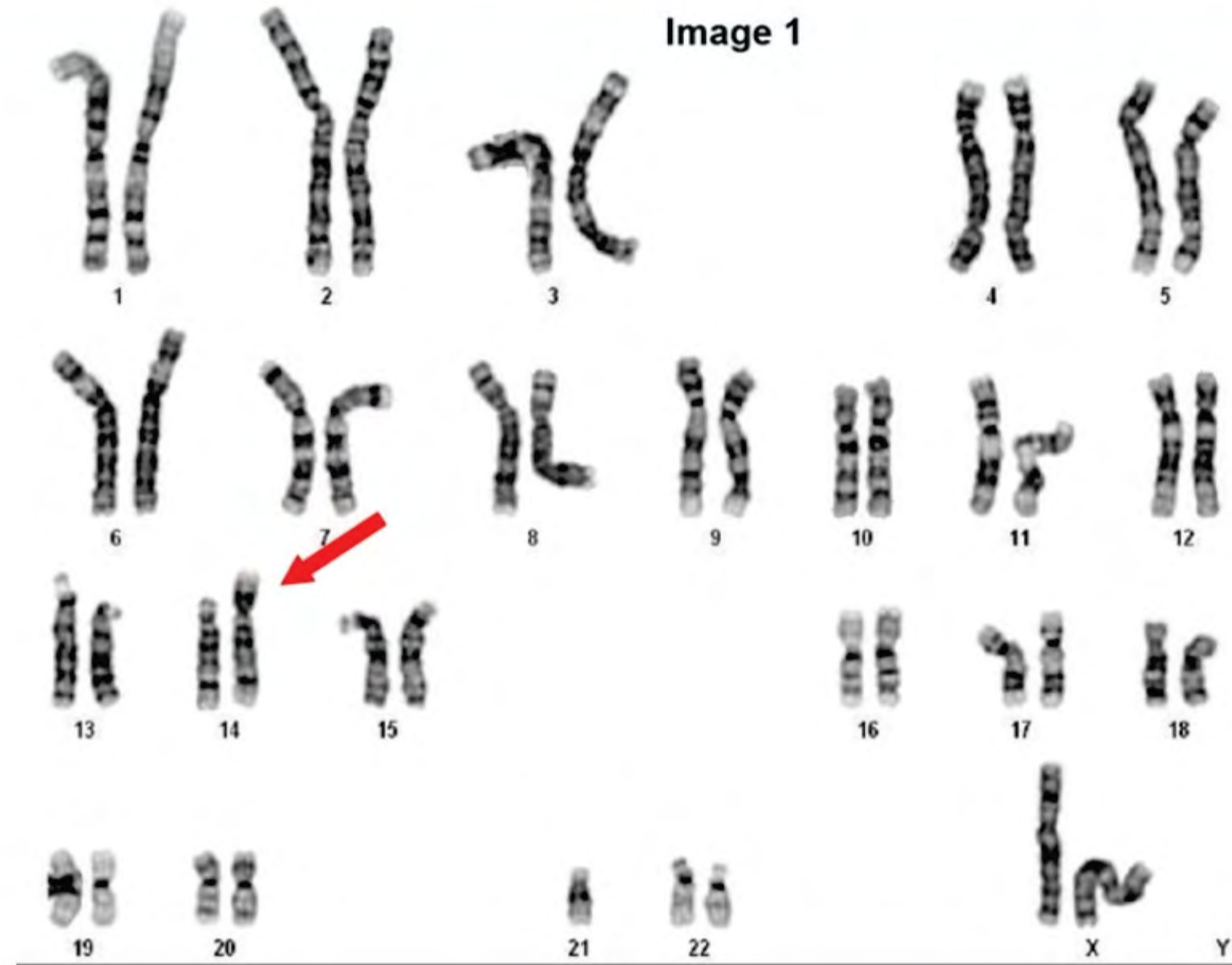


Image 1



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Exhibit Display



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(Choice E) Trisomy 18 (47, XX, +18), or Edwards syndrome, usually arises secondary to meiotic nondisjunction

A 29-year-old woman is brought to the emergency department after a syncopal episode while getting out of bed. Yesterday, the patient developed right-sided abdominal pain, which has spread across the lower abdomen over the past few hours. She also noticed a blood stain on her underwear after the syncopal episode. Her last menstrual period was 8 weeks ago. The patient uses condoms intermittently for contraception. Temperature is 37 C (98.6 F), blood pressure is 90/60 mm Hg, and pulse is 125/min. The patient has diffuse lower abdominal pain with rebound tenderness and voluntary guarding. Pelvic examination shows cervical motion tenderness and right-sided adnexal tenderness; no masses are palpated. Which of the following is the most likely diagnosis for this patient?

- ☐

A. Acute appendicitis
- ☐

B. Ovarian torsion
- ☐

C. Pelvic inflammatory disease
- ☐

D. Ruptured ectopic pregnancy
- ☐

E. Tuboovarian abscess

Submit

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- A. Acute appendicitis (0%)
- ✗

☒ B. Ovarian torsion (4%)
- C. Pelvic inflammatory disease (6%)
- ✓

☐ D. Ruptured ectopic pregnancy (87%)
- E. Tuboovarian abscess (0%)

Incorrect

Correct answer
D

87%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation

Ectopic pregnancy with ruptured fallopian tube



A 29-year-old woman is brought to the emergency department after a syncopal episode while getting out of bed.

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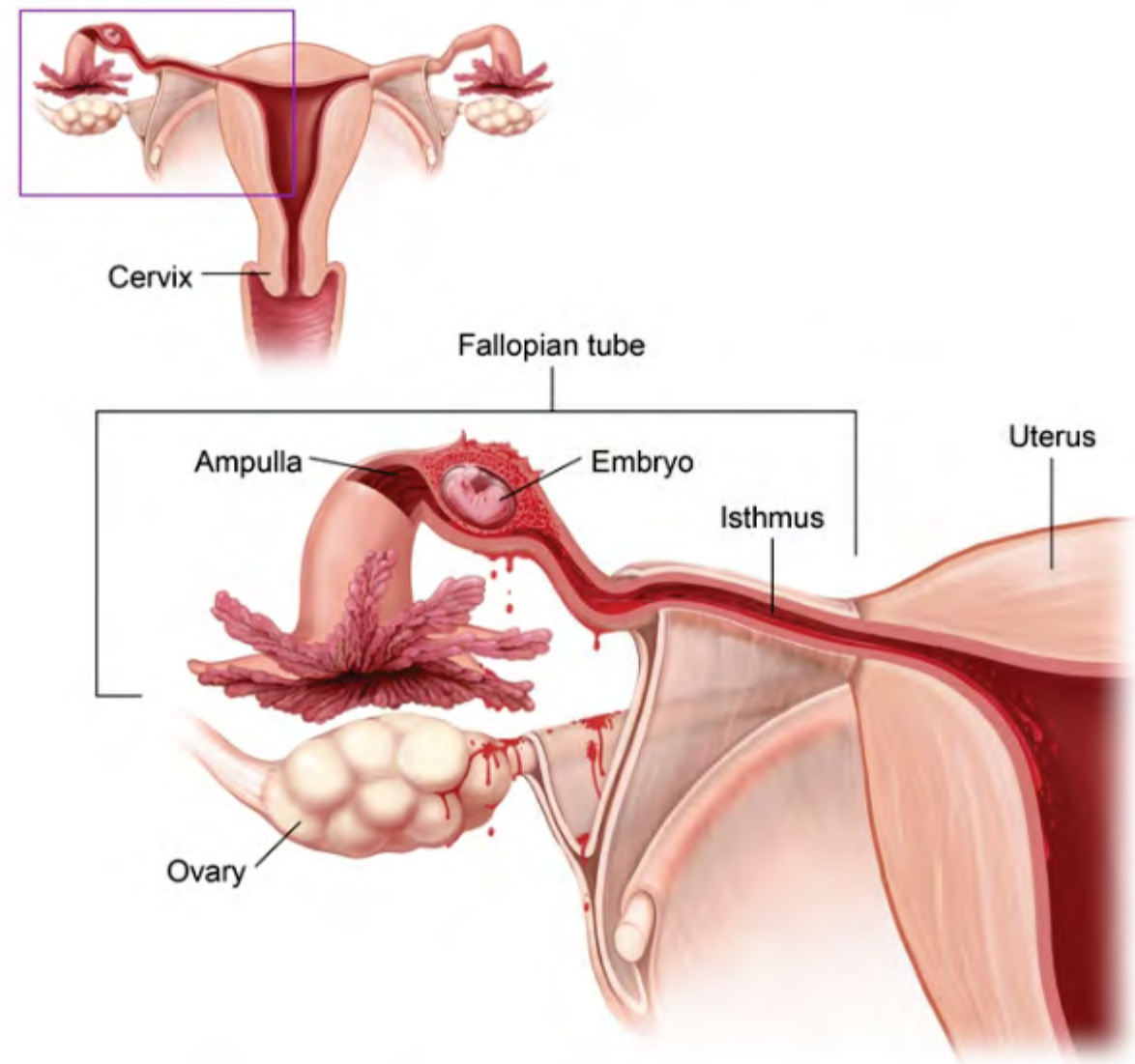
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Ectopic pregnancy with ruptured fallopian tube



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This patient with diffuse abdominal pain, rebound tenderness with guarding, and hemodynamic instability (eg, hypotension, tachycardia, syncope) likely has **intraabdominal bleeding** (ie, hemoperitoneum). In the setting of a **missed menstrual cycle** (ie, last menstrual period 8 weeks ago), **right adnexal tenderness**, and vaginal bleeding, the most likely cause is a **ruptured ectopic pregnancy**.

Ectopic pregnancies occur due to implantation in an **extrauterine location**, most commonly the fallopian tube. Unlike the uterus, which has a thick wall of distensible smooth muscle, the fallopian tube cannot expand as the ectopic pregnancy enlarges. This strain on the tubal wall, in addition to surrounding tissue ischemia (from the redirection of blood flow toward the pregnancy), causes unilateral abdominal pain and tubal rupture. Rupture at this highly vascular site typically results in brisk intraabdominal bleeding.

Because blood is highly irritating to the peritoneum, patients with **hemoperitoneum** usually develop diffuse abdominal pain with rebound and guarding; low-grade fever (due to inflammation) is also common. Patients with blood accumulation in the posterior cul-de-sac can also have cervical motion tenderness and a strong urge to defecate. Patients with hemodynamic instability (eg, orthostatic hypotension, syncopal episode) require urgent surgical management.

(Choice A) Acute appendicitis can present with right lower abdominal pain that progresses to diffuse pain in cases of appendiceal rupture (ie, peritonitis from spilled intestinal contents). However, this diagnosis is less likely in this patient who also has a missed menstrual cycle and vaginal bleeding.

(Choice B) **Ovarian torsion** can cause unilateral abdominal pain and adnexal tenderness; patients with prolonged torsion may develop rebound and guarding due to ovarian necrosis. However, the pain is typically sudden onset (ie, acute occlusion of ovarian blood flow) and vaginal bleeding is uncommon. An adnexal mass (not present in this patient) may be palpable.

(Choices C and E) Pelvic inflammatory disease (PID) typically presents with abdominal pain and cervical motion tenderness due to infection and inflammation of the upper genital tract. Tuboovarian abscess (TOA) is a severe form of PID that has localized to the adnexa. Unlike this patient, those with PID or TOA are typically febrile with

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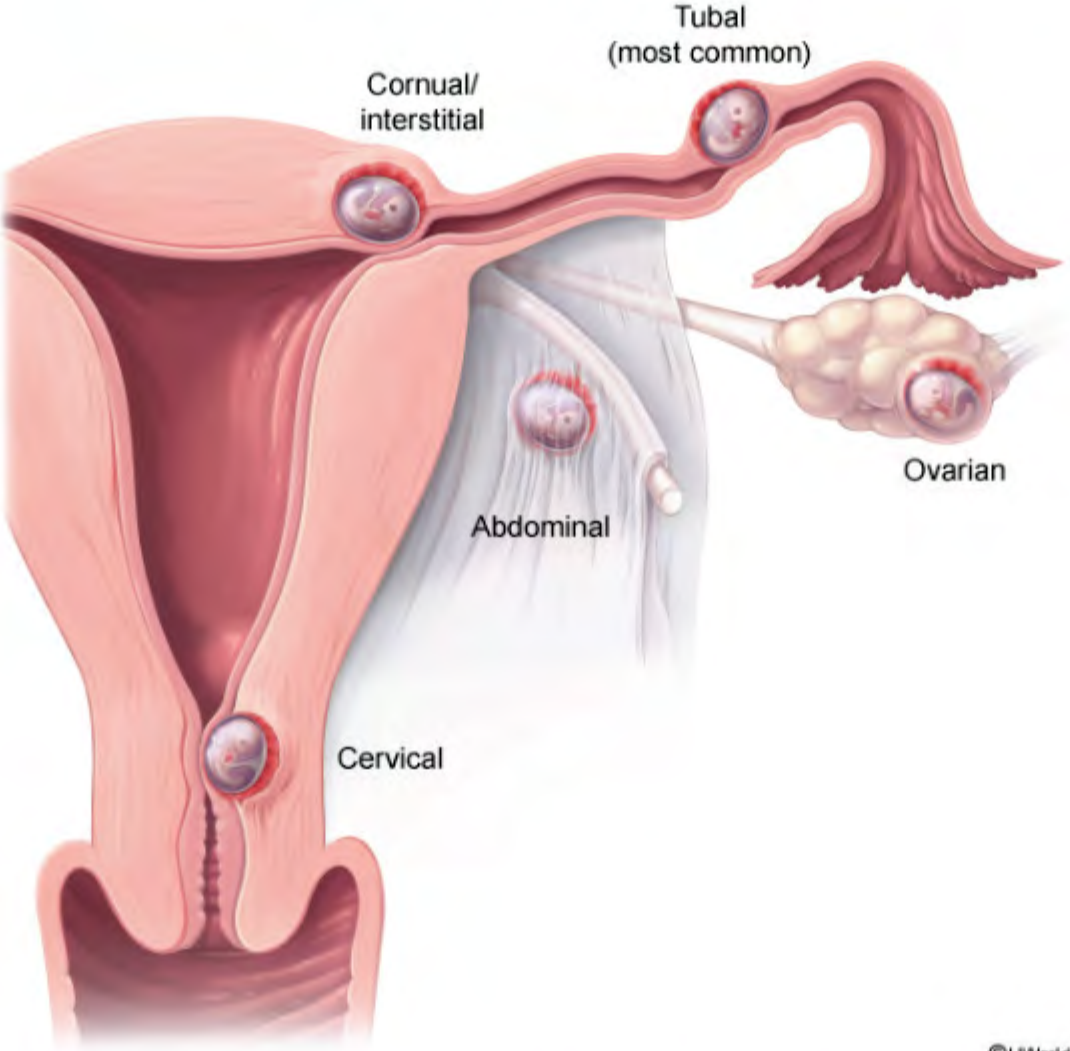
(Choices C and E) Pelvic inflammatory disease (PID) typically presents with abdominal pain and cervical motion tenderness due to infection and inflammation of the upper genital tract. Tuboovarian abscess (TOA) is a severe form of PID that has localized to the adnexa. Unlike this patient, those with PID or TOA are typically febrile with malodorous cervical discharge.

Educational objective:

Ectopic pregnancy may present with unilateral lower abdominal pain, adnexal tenderness, and vaginal bleeding. Patients with a ruptured ectopic pregnancy have severe intraabdominal bleeding, which causes diffuse abdominal pain with rebound/guarding and hemodynamic instability.

Exhibit Display

Ectopic pregnancy locations



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Exhibit Display

Ovarian torsion

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A 26-year-old woman comes to the emergency department for pelvic pain and vaginal bleeding over the past 2 days. Today, the pain has increased, but she has had no nausea, vomiting, or orthostasis. The patient was treated for chlamydia cervicitis 3 years ago. She takes no medications and has no known drug allergies. On pelvic examination, the uterus is small and mobile and there is left adnexal tenderness. Pelvic ultrasound reveals a complex, 2-cm left adnexal mass with a gestational sac and yolk sac; there is no intrauterine pregnancy or free fluid. The patient is counseled on recommended medical treatment. Which of the following is the mechanism of action of the agent of choice for this patient?

- ☐ A. Blocks progesterone receptors, resulting in uterine contractions
- ☐ B. Decreases LH secretion, causing endometrial thinning
- ☐ C. Induces prostaglandin-mediated uterine contractions
- ☐ D. Inhibits DNA synthesis to destroy actively proliferating fetal cells
- ☐ E. Produces local inflammation to impair implantation

Submit

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- A. Blocks progesterone receptors, resulting in uterine contractions (7%)
- ✗

B. Decreases LH secretion, causing endometrial thinning (3%)
- C. Induces prostaglandin-mediated uterine contractions (13%)
- ✓

D. Inhibits DNA synthesis to destroy actively proliferating fetal cells (73%)
- E. Produces local inflammation to impair implantation (1%)

Incorrect

Correct answer
D

73%
Answered correctly

04 secs
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Explanation

Medications used for pregnancy termination	
Methotrexate	<ul style="list-style-type: none">Ectopic pregnancyFolic acid antagonist (inhibits dihydrofolate reductase)Preferentially destroys proliferating fetal cells

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Mifepristone	<ul style="list-style-type: none">AbortionPartial progesterone agonist (acts as progesterone antagonist during pregnancy)Promotes placental separation & uterine contractions
Misoprostol	<ul style="list-style-type: none">AbortionProstaglandin E1 agonistStimulates uterine contractions

This patient with pelvic pain, vaginal bleeding, and a tender left adnexal mass containing a gestational sac and yolk sac has an **ectopic pregnancy**. Ectopic pregnancies occur due to pregnancy implantation in an extrauterine location, most commonly the **fallopian tube**. Risk factors include prior sexually transmitted infection (eg, *Chlamydia trachomatis*), prior abdominal surgery, and prior ectopic pregnancy.

Medical treatment for an early, unruptured ectopic pregnancy is with **methotrexate**, a **folic acid antagonist**. Methotrexate works by directly competing with folic acid to bind dihydrofolate reductase, thereby inhibiting the formation of tetrahydrofolate. This decrease in tetrahydrofolate, an integral precursor to purine and pyrimidine synthesis, **inhibits DNA synthesis** and cell reproduction. The result is **preferential destruction of actively proliferating cells**, such as **fetal cells and trophoblasts**.

(Choice A) During pregnancy, the uterus increases its number of progesterone receptors so that circulating progesterone can stimulate the endometrial and myometrial growth required for pregnancy implantation and development. Mifepristone, which acts as a progesterone antagonist when progesterone levels are high, is used for medical abortion because it causes necrosis of the uterine decidua and stimulates uterine contractions.

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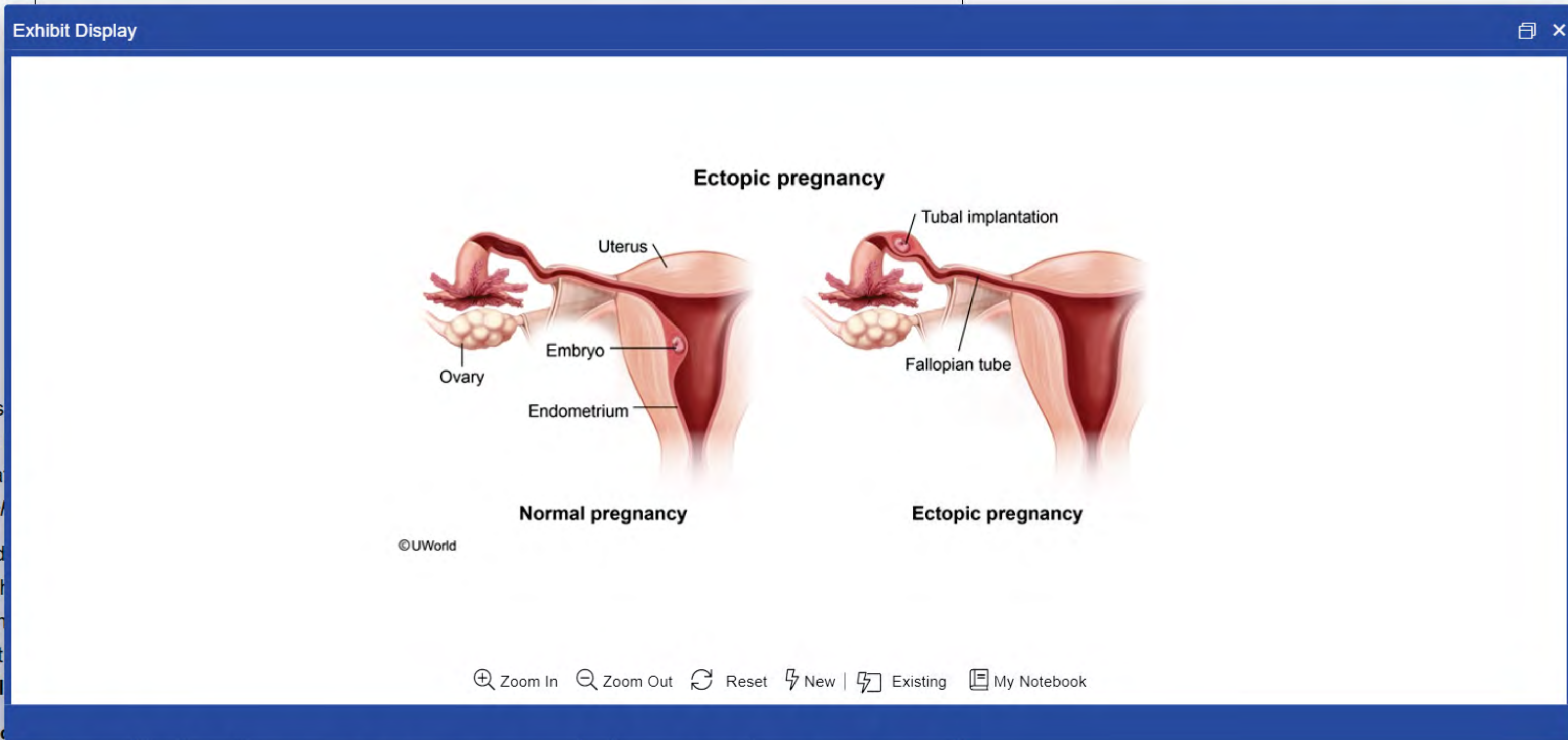
(Choice B) Progestins (eg, levonorgestrel) thin the endometrial lining and decrease LH secretion, thereby inhibiting ovulation and preventing pregnancy. They are not used in ectopic pregnancy management.

(Choice C) Misoprostol is a synthetic prostaglandin E1 agonist that acts on the uterus to induce contractions; it can be used for labor induction or medical abortion.

(Choice E) **Copper-containing intrauterine devices** produce local inflammation to impair intrauterine implantation. They are not used to treat ectopic pregnancies, which have implanted outside the uterus.

Educational objective:

Ectopic pregnancy occurs due to pregnancy implantation in an extrauterine location (eg, fallopian tube). Medical treatment is with methotrexate, which inhibits DNA synthesis and cell reproduction, primarily in actively proliferating cells such as fetal cells and trophoblasts.

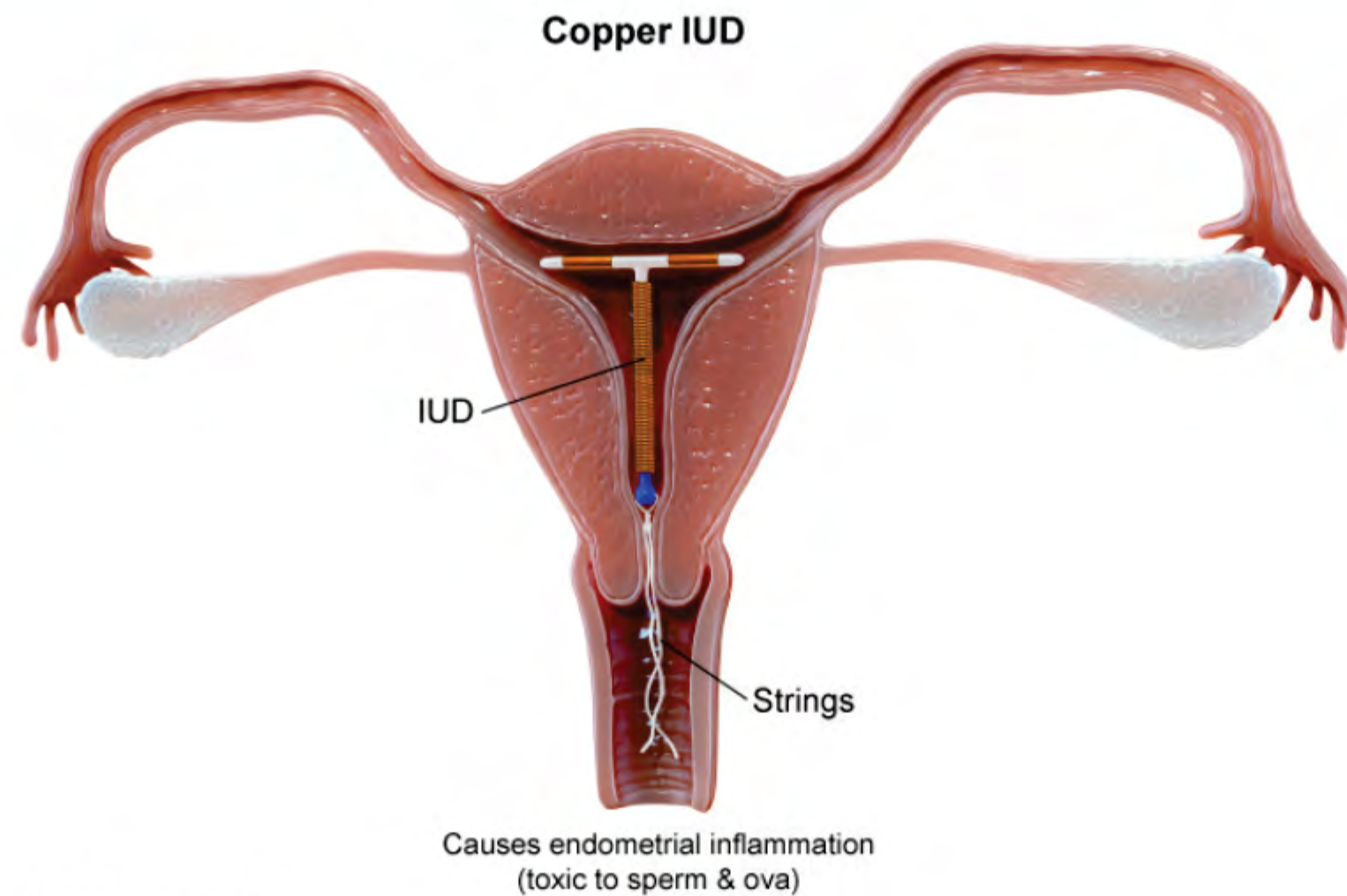


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progesterone can stimulate the endometrial and myometrial growth required for pregnancy implantation and development. Mifepristone, which acts as a progesterone antagonist when progesterone levels are high, is used for medical abortion because it causes necrosis of the uterine decidua and stimulates uterine contractions.

- Abortion

Exhibit Display



IUD = intrauterine device.
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Educational objective:



A 35-year-old woman, gravida 1 para 0, at 40 weeks gestation undergoes a cesarean delivery. Shortly after delivery of the infant, the patient becomes anxious, short of breath, and then unresponsive. Blood pressure is 70/40 mm Hg and pulse is 120/min. Oxygen saturation is 83% on room air. There is profuse bleeding from the abdominal incision and intravenous lines. The lungs are clear to auscultation. Intraarterial blood pressure monitoring is established, and pulmonary artery catheterization is performed. Initial measurements are as follows:

Central venous pressure	14 mm Hg (normal: 6-8)
Pulmonary capillary wedge pressure	5 mm Hg (normal: 6-12)

Which of the following additional findings is most likely to be seen in this patient?

- ☐ A. Decreased right ventricular preload

☐ B. Increased cardiac index

☐ C. Increased left atrial pressure

☐ D. Increased pulmonary vascular resistance

Submit

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Central venous pressure	14 mm Hg (normal: 6-8)
Pulmonary capillary wedge pressure	5 mm Hg (normal: 6-12)

Which of the following additional findings is most likely to be seen in this patient?

- A. Decreased right ventricular preload (28%)

X

B. Increased cardiac index (10%)

C. Increased left atrial pressure (4%)

D. Increased pulmonary vascular resistance (56%)

Incorrect

Correct answer D

56%

Answered correctly

03 secs

Time Spent

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Explanation

This newly postpartum patient most likely has **amniotic fluid embolus syndrome** (AFES), which is a rare but catastrophic obstetric emergency. AFES occurs when amniotic fluid enters the maternal circulation through sites of maternofetal connections (eg, endocervical veins, uterine incisions) and initiates an anaphylactoid reaction with the widespread release of proinflammatory and vasoactive substances, resulting in **cardiovascular collapse**.

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The hemodynamic effects of AFES are complex, but the primary disturbance is typically within the pulmonary vasculature, with marked elevation in pulmonary pressures due to potent **pulmonary arterial vasoconstriction** and possible vascular obstruction by cellular and acellular debris; the hemodynamic effects of this **increased pulmonary vascular resistance** are most consistent with [obstructive shock](#).

Impeded cardiopulmonary blood flow leads to elevated central venous pressure, which is a reflection of right atrial pressure and right ventricular preload (**Choice A**). The decreased forward flow leads to reduced pulmonary capillary wedge pressure, which is a reflection of left atrial pressure and representative of left ventricular preload (**Choice C**). As less blood is pumped to the left side of the heart, the cardiac index decreases, resulting in hypotension (**Choice B**). In response, systemic vascular resistance is increased in an attempt to maintain adequate tissue perfusion pressure.

AFES also commonly involves a **consumptive coagulopathy** (ie, disseminated intravascular coagulation), which manifests clinically with uterine hemorrhage and bleeding from incisions and intravenous lines.

Educational objective:

Amniotic fluid embolus syndrome is a rare but catastrophic obstetric emergency that leads to respiratory failure and obstructive shock with an elevated central venous pressure and a decreased pulmonary capillary wedge pressure and cardiac index. It also commonly involves a consumptive coagulopathy with widespread hemorrhage.

References

- [Amniotic fluid embolism](#).

CO = cardiac output; CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; PE = pulmonary embolism; PTX = pneumothorax; SVR = systemic vascular resistance. ©UWorld

A 19-year-old woman comes to the emergency department with pelvic pain, fever, and chills that began last night. The patient's last menstrual period was 12 weeks ago, and she had a surgical pregnancy termination at a clinic 2 days ago. Temperature is 38.3 C (100.9 F), blood pressure is 92/60 mm Hg, and pulse is 102/min. Physical examination shows diffuse lower abdominal tenderness without rebound or guarding. Speculum examination reveals an open cervical os and malodorous tissue in the vaginal canal. Bimanual examination shows moderate uterine tenderness and no adnexal masses. Which of the following is the most likely cause of this patient's condition?

- ☐ A. *Actinomyces* species
- ☐ B. *Chlamydia trachomatis*
- ☐ C. *Lactobacilli* species
- ☐ D. *Listeria monocytogenes*
- ☐ E. *Staphylococcus aureus*

Submit

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- A. *Actinomyces* species (12%)
- ✗

B. *Chlamydia trachomatis* (22%)
- C. *Lactobacilli* species (11%)
- D. *Listeria monocytogenes* (5%)
- ✓

E. *Staphylococcus aureus* (47%)

Incorrect

Correct answer
E

47%
Answered correctly

03 secs
Time Spent

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Version

Explanation

Septic abortion	
Risk factors	<ul style="list-style-type: none">Pregnancy termination with retained POCs
	<ul style="list-style-type: none">Enterobacteriaceae

Septic abortion	
Risk factors	<ul style="list-style-type: none">• Pregnancy termination with retained POCs
Microbiology	<ul style="list-style-type: none">• Enterobacteriaceae• Group A <i>Streptococcus</i>• <i>Staphylococcus aureus</i>
Clinical presentation	<ul style="list-style-type: none">• Fever, lower abdominal pain• Malodorous, purulent cervical discharge• Tender uterus with dilated cervix
POCs = products of conception.	

This patient with **fever**, abdominal pain, **uterine tenderness**, and a **malodorous cervical discharge** most likely has a **septic abortion**. With [surgical pregnancy termination](#), the cervix is dilated, and the products of conception (POCs) are removed via suction and sharp curettage. During this procedure, vaginal flora and other bacteria can enter the uterine cavity; complete removal of the POCs, including placental and fetal tissue, does not usually promote bacterial growth. However, when incomplete evacuation occurs, the **retained POCs** in the uterus can become **infected** and result in a septic abortion.

The most common organisms that cause septic abortions include **anaerobes** (eg, Enterobacteriaceae), **group A *Streptococcus***, and ***Staphylococcus aureus***. Once introduced into the uterus, bacteria can access the intervillous space of the retained placenta, which leads to uterine infection and bacteremia. Bacterial toxins can cause tissue necrosis, widespread endothelial damage, multiorgan failure, and even death.

In addition to **broad-spectrum antibiotics**, patients require **urgent surgical uterine evacuation** to remove the necrotic nidus of infection, where the antibiotics are less effective. Long-term complications include synechiae (adhesions) in the uterine cavity that can lead to secondary amenorrhea and infertility (Asherman syndrome).

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(Choice A) *Actinomyces* species normally colonize the mouth and digestive and genital tracts. They can cause clinically significant pelvic infections in women using intrauterine devices, but they are not typically involved in septic abortions.

(Choice B) *Chlamydia trachomatis* can cause **cervicitis** and pelvic inflammatory disease characterized by pelvic pain with fever and cervical motion tenderness. This pathogen is not typically involved in septic abortion because it is not a vaginal colonizer.

(Choice C) *Lactobacillus* is found within the vaginal microbiota and prevents the overgrowth of pathogens (anaerobes) through the production of hydrogen peroxide. In immunocompetent hosts, lactobacilli do not lead to invasive infection.

(Choice D) *Listeria monocytogenes* is a food-borne pathogen that is especially virulent in immunocompromised

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(Choice D) *Listeria monocytogenes* is a food-borne pathogen that is especially virulent in immunocompromised individuals and pregnant women. It causes spontaneous abortion, intrauterine fetal infection, and/or neonatal sepsis through transplacental transmission.

Educational objective:

Symptoms of septic abortion typically include fever, abdominal pain, uterine tenderness, and/or malodorous discharge after pregnancy termination. Common pathogens include Enterobacteriaceae, group A *Streptococcus*, and *Staphylococcus aureus*. Treatment involves urgent surgical evacuation and broad-spectrum antibiotics.

presentation

- Tender uterus with dilated cervix

Exhibit Display

Suction curettage

The diagram illustrates the suction curettage procedure. A speculum is shown inserted into the vagina to visualize the cervix. A suction curette, which is a long, thin tube with a small cup at the end, is inserted into the uterus. The cup is positioned against the uterine wall, and suction is applied to remove the pregnancy tissue. Labels point to the 'Pregnancy' (the tissue being removed), the 'Suction curette' (the instrument), the 'Uterus' (the organ), and the 'Speculum' (the instrument used to visualize the cervix).

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This patient with **fever**, abdominal pain, **uterine tenderness**, and a **malodorous cervical discharge** most likely

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Exhibit Display

Cervicitis

Normal cervix

Red, inflamed, friable cervix

Purulent discharge

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(Choice D) *Listeria monocytogenes* is a food-borne pathogen that is especially virulent in immunocompromised individuals and pregnant women. It causes spontaneous abortion, intrauterine fetal infection, and/or neonatal

An 18-year-old woman comes to the office for evaluation of acne. The patient is very upset about the acne scarring, which worsened since she started college. She has been very stressed by upcoming examinations and feels "exhausted" due to lack of adequate sleep. The patient has been using topical benzoyl peroxide and tretinoin for the past year. She was also prescribed an oral antibiotic but stopped taking it several months ago due to lack of improvement. The patient is sexually active with her boyfriend and uses an intrauterine device for contraception. Her periods are regular and last 5 days. She takes no other medications and does not use tobacco, alcohol, or illicit drugs. Physical examination shows nodulocystic acne with scarring on the face, chin, and upper back. The patient is interested in isotretinoin treatment. Which of the following is the best next step in management of this patient?

- ☐ A. Bone density test
- ☐ B. Pelvic ultrasound
- ☐ C. Removal of intrauterine device
- ☐ D. Serum retinol level
- ☐ E. Serum testosterone
- ☐ F. Serum TSH level
- ☐ G. Urine β -hCG

Submit

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- A. Bone density test (0%)

✗

B. Pelvic ultrasound (2%)

C. Removal of intrauterine device (2%)

D. Serum retinol level (4%)

E. Serum testosterone (4%)

F. Serum TSH level (1%)

✓

G. Urine β-hCG (84%)

Incorrect

Correct answer
G

84%
Answered correctly

04 secs
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Explanation

Adverse effects of oral isotretinoin therapy
<ul style="list-style-type: none">• Teratogenic (eg, spontaneous abortion, fetal malformations)• Hyperlipidemia• Chelitis, dry skin• Myalgias• Pseudotumor cerebri

Isotretinoin is a synthetic 13-*cis*-isomer of naturally occurring all-*trans*-retinoic acid (tretinoin), a derivative of vitamin A. It can be administered orally to treat severe acne associated with significant **scarring**. It is also indicated in patients who fail therapy with topical tretinoin/benzoyl peroxide and antibiotics. **Retinoids** inhibit follicular epidermal keratinization, thereby loosening the keratin plugs of comedones and facilitating their expulsion. They also reduce the size of sebaceous glands and inhibit sebum production.

Isotretinoin is a potent teratogen and is absolutely **contraindicated in pregnancy**. Exposure to this medication can cause spontaneous abortion and a variety of severe fetal anomalies. A **negative pregnancy test** (urine or serum) is required prior to prescribing, and abstinence/contraception is recommended. Monthly pregnancy testing is required during treatment. An intrauterine device (IUD) is a long-term contraception method that is 99% effective and safe to use in women of all ages. It should not be discontinued in this patient (**Choice C**). Because no contraception method is 100% effective, condom use as a backup method is advised.

(Choice A) Bone density testing is recommended in patients on long-term glucocorticoid therapy because these drugs increase bone resorption and reduce bone formation. Skeletal complications from isotretinoin therapy are rare.

(Choices B and E) Pelvic ultrasound and serum testosterone are used to diagnose polycystic ovary syndrome (PCOS), a major underlying cause of acne. PCOS commonly presents with oligomenorrhea and hirsutism, which are absent in this patient. Pelvic ultrasound often reveals ovaries with multiple small cysts. Treatment with oral contraceptives restores menses and alleviates acne.

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(Choice D) Vitamin A deficiency is rare in developed countries. Unlike vitamin A, isotretinoin is not stored in the liver and is therefore not associated with toxicities seen with high-dose vitamin A intake. Serum retinol measurement is not indicated.

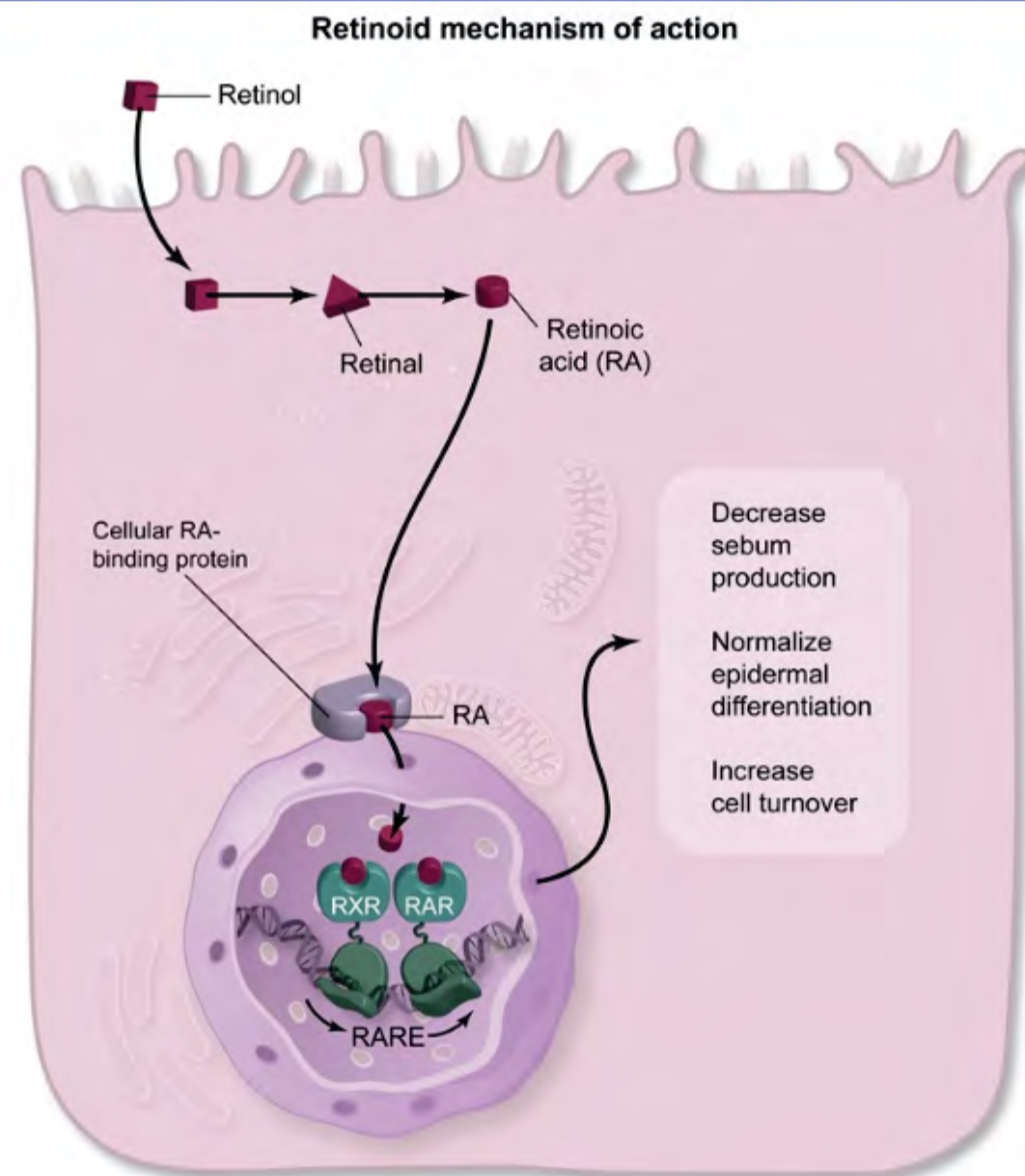
(Choice F) Hypothyroidism commonly causes fatigue in addition to cold intolerance and constipation, but it does not cause acne. In addition, serum TSH would be elevated in a patient with hypothyroidism.

Educational objective:

Isotretinoin is used to treat severe acne with significant scarring. Pregnancy is an absolute contraindication due to the risk of teratogenicity. Sexually active women should be advised to use 2 forms of contraception and take monthly pregnancy tests.

- Teratogenic (eg, spontaneous abortion, fetal malformations)

Exhibit Display



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(Choice D) Vitamin A deficiency is rare in developed countries. Unlike vitamin A, isotretinoin is not stored in the

A 38-year-old woman, gravida 1 para 0, at 32 weeks gestation comes to the office due to a progressively worsening headache. The patient also reports increased leg swelling and a 4.54-kg (10-lb) weight gain since her last visit 2 weeks ago. She has no chronic medical conditions, and her pregnancy has been uncomplicated. Review of medical records shows that blood pressure was 130/80 mm Hg at 30 weeks gestation. Today, blood pressure is 165/100 mm Hg. There is 2+ pitting edema to the knees. Serum creatinine is 0.9 mg/dL. Which of the following findings would most likely be present in this patient's urinalysis?

	Specific gravity	Protein	Red blood cells	
<input type="radio"/> A.	Decreased	None	None	(4%)
<div>X</div> <input checked="" type="radio"/> B.	Decreased	+	None	(16%)
<input type="radio"/> C.	Increased	None	+	(1%)
<div>✓</div> <input type="radio"/> D.	Increased	+	None	(67%)
<input type="radio"/> E.	Normal	None	+	(0%)
<input type="radio"/> F.	Normal	+	+	(8%)

Incorrect

Correct answer D

67% Answered correctly

04 secs Time Spent

2023 Version

Explanation

Hypertensive disorders of pregnancy

Hypertensive disorders of pregnancy	
Chronic hypertension	<ul style="list-style-type: none">Systolic pressure ≥140 mm Hg &/or diastolic pressure ≥90 mm Hg prior to conception or at <20 weeks gestation
Gestational hypertension	<ul style="list-style-type: none">New-onset elevated blood pressure at ≥20 weeks gestationNo proteinuria or signs of end-organ damage
Preeclampsia	<ul style="list-style-type: none">New-onset elevated blood pressure at ≥20 weeks gestation AND <ul style="list-style-type: none">Proteinuria OR signs of end-organ damage
Eclampsia	<ul style="list-style-type: none">Preeclampsia AND <ul style="list-style-type: none">New-onset tonic-clonic seizures

This patient with a worsening headache and **new-onset hypertension** (ie, systolic ≥140 or diastolic ≥90 mm Hg) **after 20 weeks gestation** has preeclampsia. **Preeclampsia** is most likely caused by narrowed uteroplacental spiral artery formation and abnormal placentation, which lead to placental hypoperfusion and ischemia. These changes trigger the release of antiangiogenic factors that cause widespread maternal vasoconstriction (eg, hypertension), endothelial cell damage (eg, third spacing with edema and weight gain), and **end-organ damage** (eg, headache).

The kidneys are commonly affected by preeclampsia, and **urinalysis** can support the diagnosis:

- Third spacing (ie, intravascular volume depletion) and vasoconstriction of the renal vessels result in decreased urine production (ie, oliguria) and concentrated urine (ie, **increased specific gravity**) because the kidneys attempt to retain sodium and water.
- Damage to the renal endothelium increases glomerular permeability and allows for leakage of large

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- Damage to the renal endothelium increases glomerular permeability and allows for leakage of large molecules, as evidenced by **proteinuria**, which is classic for preeclampsia.
- Renal vasoconstriction causes a decreased glomerular filtration rate (GFR) and an **increased serum creatinine level** (above baseline). Healthy pregnant patients have decreased baseline serum creatinine levels due to blood volume expansion and increased GFR; therefore, a normal-appearing creatinine level (0.7-0.9 mg/dL) during pregnancy, as seen in this patient, typically indicates renal compromise.

Definitive management of preeclampsia is with delivery, and renal function typically recovers postpartum.

(Choices A and B) Decreased urine specific gravity (ie, decreased concentration of urine solutes) is associated with excessive fluid intake (eg, primary polydipsia) or diabetes insipidus. It is not associated with hypertension in pregnancy.

(Choices C, E, and F) Microscopic hematuria (ie, red blood cells on urinalysis) is typically associated with urinary tract disease, such as infection, kidney or bladder cancer, and renal calculus. Hematuria is not associated with hypertension in pregnancy.

(eg, headache).

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Educational objective:

Preeclampsia is new-onset hypertension after 20 weeks gestation with signs of end-organ damage (eg, headache). Renal effects of preeclampsia include oliguria with increased specific gravity, proteinuria, and serum creatinine levels.

A 28-year-old woman, gravida 3 para 2, at 31 weeks gestation comes to the office for a prenatal visit. The patient has had increasing shortness of breath while climbing stairs and during long walks for the past few weeks but has had no cough, sick contacts, hemoptysis, orthopnea, or chest pain. She has had no contractions or vaginal bleeding, and fetal movement is normal. The patient has no chronic medical conditions, and her pregnancy has been uncomplicated. Blood pressure is 110/80 mm Hg, pulse is 70/min, and respirations are 18/min. Fetal heart tones are normal. Cardiac examination reveals no rubs or murmurs. The lungs are clear to auscultation bilaterally. The remainder of the examination is unremarkable. Which of the following laboratory findings will most likely be found in this patient?

	pH	PaCO ₂ (mm Hg)	PaO ₂ (mm Hg)	Urine bicarbonate
<input type="radio"/> A.	7.26	45	101	↓
<input type="radio"/> B.	7.34	30	97	↑
<input type="radio"/> C.	7.46	29	102	↑
<input type="radio"/> D.	7.48	44	98	↓
<input type="radio"/> E.	7.60	25	94	↓

Submit

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<input type="radio"/> A.	7.26	45	101	↓	(14%)
<input checked="" type="radio"/> B.	7.34	30	97	↑	(21%)
<input type="radio"/> C.	7.46	29	102	↑	(38%)
<input type="radio"/> D.	7.48	44	98	↓	(16%)
<input type="radio"/> E.	7.60	25	94	↓	(8%)

Incorrect

Correct answer C

38% Answered correctly

04 secs Time Spent

2023 Version

Explanation

A.

7.26

45

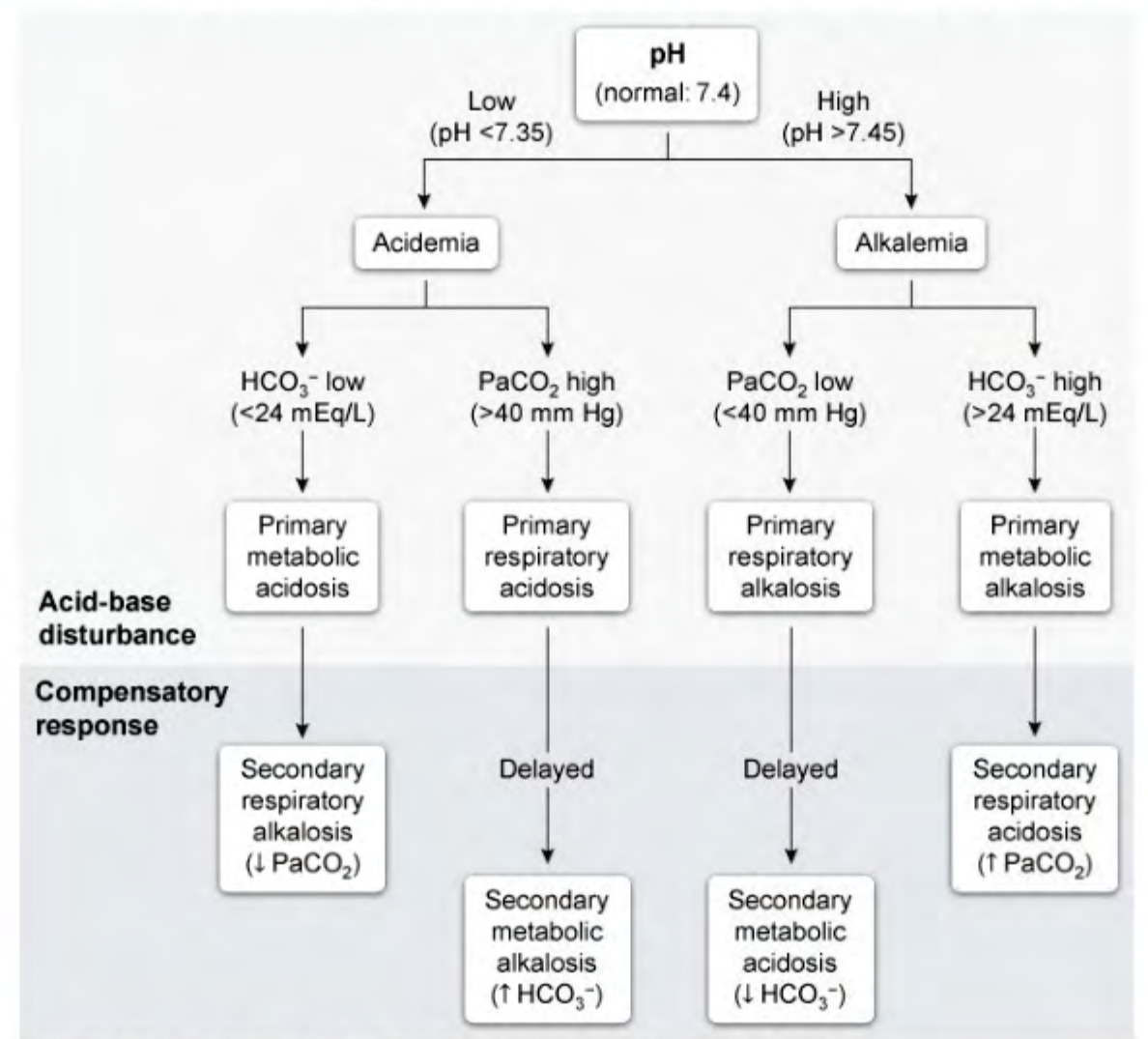
101

↓

(14%)

Exhibit Display

Arterial blood gas interpretation of acid-base disorders



* The normal ranges for PaCO₂ and HCO₃⁻ vary slightly around 40 mm Hg and 24 mEq/L. For simplicity, these numbers should be used as a normal baseline for acid-base calculations.
HCO₃⁻ = bicarbonate; PaCO₂ = partial pressure of carbon dioxide in arterial blood.

The **normal physiology of pregnancy** involves **chronic hyperventilation**. Elevated **progesterone** levels trigger a sensation of **shortness of breath** and stimulate the hypothalamus to increase respiratory drive. Ventilation is increased primarily via **increased tidal volume**, which is facilitated by increased diaphragmatic excursion and hormone-induced laxity of the intercostal muscles that allows for enlargement of the thoracic cavity (although the expanding uterus displaces the resting position of the diaphragm upward, diaphragmatic excursion is not impaired). A slight increase in respiratory rate also contributes.

The hyperventilation of pregnancy creates an expected **respiratory alkalosis** (ie, pH >7.45 and partial pressure of carbon dioxide in arterial blood [PaCO₂] <40 mm Hg) with metabolic compensation (**increased renal bicarbonate [HCO₃⁻] excretion**). Arterial oxygenation is also increased slightly above normal (ie, arterial partial pressure of oxygen [PaO₂] >100 mm Hg). These changes help facilitate the transport of acidic wastes from, and the transport of oxygen to, the developing fetus.

(Choice A) Low pH (<7.35) with elevated PaCO₂ (>40 mm Hg) indicates respiratory acidosis. Urine HCO₃⁻ is low as the kidneys increase HCO₃⁻ reabsorption for metabolic compensation.

(Choice B) Low pH is not explained by low PaCO₂ (<40 mm Hg); therefore, the low PaCO₂ likely represents respiratory compensation for metabolic acidosis. Renal loss of HCO₃⁻ (ie, high urine HCO₃⁻), as occurs with renal tubular acidosis, is a common cause of metabolic acidosis.

(Choice D) High pH (>7.45) is not explained by elevated PaCO₂; therefore, the elevated PaCO₂ likely represents respiratory compensation for metabolic alkalosis. Renal retention of HCO₃⁻ (ie, low urine HCO₃⁻), as occurs with severe vomiting and diuretic overuse, commonly contributes to metabolic alkalosis.

(Choice E) High pH with low PaCO₂ indicates respiratory alkalosis. Urine HCO₃⁻ may be low in the acute setting but begins to increase after several hours to provide metabolic compensation (full metabolic compensation requires approximately 72 hours). This patient at 31 weeks gestation will have fully compensated respiratory alkalosis with increased urine HCO₃⁻ excretion.

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Educational objective:

The normal physiology of pregnancy involves chronic hyperventilation caused by elevated progesterone levels stimulating an increase in central respiratory drive. This creates an expected respiratory alkalosis with metabolic compensation (increased renal bicarbonate excretion) that helps facilitate the transport of acidic wastes from, and the transport of oxygen to, the developing fetus.

A 24-year-old woman, gravida 1 para 0, at 28 weeks gestation comes to the office for a routine prenatal visit. She feels well and has had no vaginal bleeding, contractions, or leakage of fluid. Fetal movement is normal. The patient has no chronic medical conditions and her pregnancy has been uncomplicated. She previously had heavy menstrual periods that were managed with oral contraceptives. Blood pressure is 126/84 mm Hg and pulse is 78/min. Fundal height and fetal heart tones are normal. Today's third-trimester complete blood count is as follows:

Hemoglobin	11.2 g/dL
Platelets	120,000/mm ³
Leukocytes	8,200/mm ³

Coagulation studies and peripheral smear are within normal limits. The patient's first-trimester platelet count was 160,000/mm³. Urine dipstick today is negative for protein. Which of the following is the most likely cause of this patient's thrombocytopenia?

- ☐

A. Coagulation cascade—activated thrombi formation
- ☐

B. Decreased thromboxane A2 production
- ☐

C. Hemodilution and increased platelet sequestration
- ☐

D. Immune complex—mediated complement activation
- ☐

E. Small vessel formation of platelet microthrombi

Submit

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- A. Coagulation cascade—activated thrombi formation (7%)

✗

B. Decreased thromboxane A2 production (4%)

✓

C. Hemodilution and increased platelet sequestration (78%)

D. Immune complex—mediated complement activation (4%)

E. Small vessel formation of platelet microthrombi (5%)

Incorrect

Correct answer
C

78%

Answered correctly

03 secs

Time Spent

2023

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Explanation

This patient at 28 weeks gestation has mild, asymptomatic thrombocytopenia most consistent with **gestational thrombocytopenia** (GT), a benign, self-limited condition that can occur during pregnancy. GT is thought to arise due to the following:

- **Plasma volume expansion:** Maternal plasma volume expands approximately 30% during pregnancy to facilitate optimal fetal nutrient delivery and protect against maternal hemorrhage during delivery. Platelet production, however, does not typically increase during pregnancy; therefore, there is **hemodilution** and resultant thrombocytopenia.
- **Sequestration/consumption of platelets** in the placenta: The placenta is a highly vascular organ with similarities to the spleen, a major site of normal platelet sequestration. Platelets pool in the placental circulation and undergo accelerated consumption within the intervillous space, thereby reducing the serum platelet count.

GT is commonly diagnosed in the **third trimester**, when plasma volume peaks; it is often an incidental finding on routine bloodwork. Patients with GT have mild thrombocytopenia (platelet count usually $\geq 100,000/\text{mm}^3$), no symptoms (eg, no bruising or bleeding), normal coagulation studies, and normal peripheral blood smear.

(Choice A) Disseminated intravascular coagulation (DIC) is characterized by widespread activation of the coagulation cascade with thrombocytopenia due to platelet consumption. However, DIC is associated with bleeding and abnormal coagulation studies, which are not seen in this patient.

(Choice B) Aspirin decreases the production of thromboxane A2 and inhibits platelet aggregation. Patients taking aspirin usually have normal or elevated platelet counts and decreased platelet clotting function.

(Choice D) Immune complex–mediated complement activation occurs in inflammatory rheumatologic diseases (eg, systemic lupus erythematosus). Although these conditions are often associated with thrombocytopenia, patients typically have other symptoms.

(Choice E) Thrombotic thrombocytopenic purpura (TTP) causes thrombocytopenia due to the formation of platelet

resultant thrombocytopenia.

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(Choice E) Thrombotic thrombocytopenic purpura (TTP) causes thrombocytopenia due to the formation of platelet clots in the microvasculature. However, TTP typically causes fever, microangiopathic hemolytic anemia, and neurologic abnormalities.

Educational objective:

Gestational thrombocytopenia is common in the third trimester and is typically mild, asymptomatic, and not associated with other laboratory abnormalities. It occurs due to hemodilution (ie, maternal plasma volume expansion) and increased sequestration/consumption of platelets in the placenta.

An 18-year-old primigravida at 27 weeks gestation comes to the emergency department due to severe right upper quadrant pain. The pain started 2 hours ago, and the patient has also had nausea, vomiting, and irregular uterine contractions. Temperature is 36.7 C (98.1 F), blood pressure is 172/118 mm Hg, and pulse is 110/min. BMI is 34 kg/m². On abdominal examination, tenderness to palpation is present over the right upper quadrant. Aspartate aminotransferase (SGOT) is 220 U/L and alanine aminotransferase (SGPT) is 240 U/L. Abdominal imaging reveals a hepatic subcapsular hematoma. This patient is most at risk for which of the following complications?

- ☐ A. Acute interstitial nephritis
- ☐ B. Bleeding esophageal varices
- ☐ C. Intraamniotic infection
- ☐ D. Pancreatitis
- ☐ E. Seizures
- ☐ F. Thrombocytosis

Submit

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- A. Acute interstitial nephritis (1%)

✗

B. Bleeding esophageal varices (11%)

C. Intraamniotic infection (6%)

D. Pancreatitis (5%)

✓

E. Seizures (55%)

F. Thrombocytosis (19%)

Incorrect

Correct answer
E

55%
Answered correctly

03 secs
Time Spent

2023
Version

Explanation

This pregnant patient with **hypertension** and right upper quadrant pain likely has **preeclampsia with severe features** (ie, new hypertension at >20 weeks gestation with signs of severe end-organ damage) and/or **HELLP** (**H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelet count) **syndrome**. Both conditions share common features and likely represent a spectrum of disease; they are characterized by widespread endothelial dysfunction that leads to

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Both vasospasm and microthrombi deposition result in decreased hepatic blood flow, liver ischemia, and **hepatocellular injury** (eg, nausea, vomiting, right upper quadrant pain, elevated transaminases). Severe cases may progress to liver necrosis and hemorrhage, causing a **subcapsular hematoma** (ie, **blood pooling** beneath the hepatic [Glisson] capsule with capsular distension).

Because preeclampsia and HELLP syndrome cause widespread endothelial dysfunction and vasospasm, multiple organ systems are affected, and patients are at increased risk for other complications. These include **seizures** (due to cerebral vasospasm, thrombosis, and/or edema); renal insufficiency (due to **intrarenal vasospasm**); thrombocytopenia; and disseminated intravascular coagulation (due to overactivation of the coagulation cascade) **(Choice F)**.

(Choice A) Although this patient is at risk for renal insufficiency, she is not at increased risk for acute interstitial nephritis, which is typically drug induced (eg, nonsteroidal anti-inflammatory drugs).

(Choice B) Bleeding esophageal varices typically occur in patients with liver cirrhosis due to venous portal hypertension. In contrast, HELLP syndrome and preeclampsia cause arterial hypertension.

(Choice C) The risk for intraamniotic infection is increased with prolonged rupture of membranes or genitourinary infection (eg, group B *Streptococcus*, *Chlamydia trachomatis*).

(Choice D) Risk factors for pancreatitis include biliary disease, chronic alcohol use, and hypertriglyceridemia.

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(Choice D) Risk factors for pancreatitis include biliary disease, chronic alcohol use, and hypertriglyceridemia.

Educational objective:

Pregnant women with severe preeclampsia can develop HELLP (Hemolysis, Elevated Liver enzymes, Low Platelet count) syndrome. Nausea/vomiting and right upper quadrant pain occur due to liver ischemia and/or hemorrhage (eg, subcapsular hematoma). These patients are also at risk for seizure, renal insufficiency, and disseminated intravascular coagulation due to widespread endothelial dysfunction.

Exhibit Display

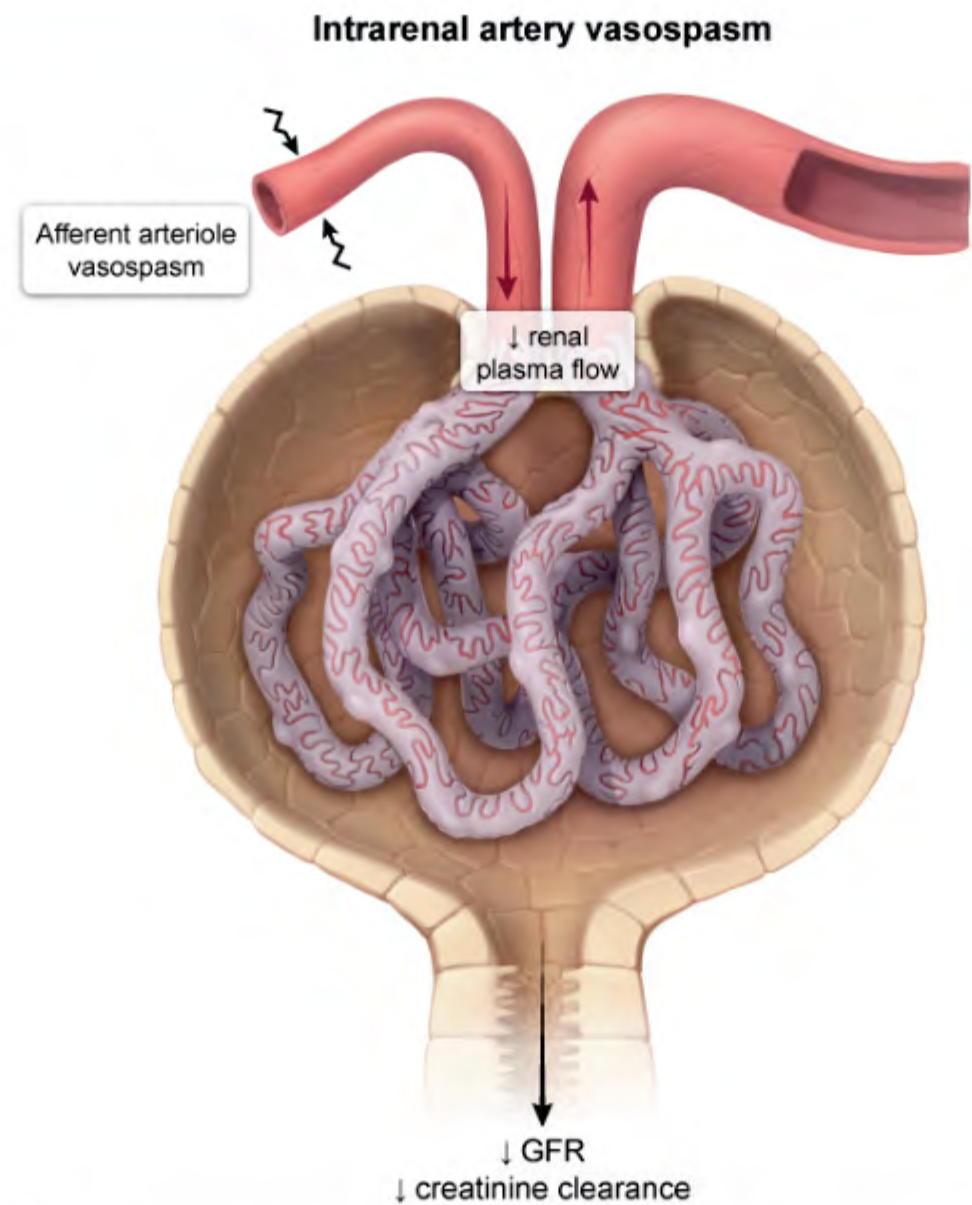
Subcapsular hematoma



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Exhibit Display



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A 33-year-old woman, gravida 2 para 1, is brought to the labor and delivery unit at 32 weeks gestation and precipitously delivers a male infant within 2 minutes of arrival. The infant appears small for gestational age. Apgar scores are 5 and 6 at 1 and 5 minutes, respectively. The mother did not receive prenatal care with this pregnancy. On delivery, the placenta appears pale and enlarged. In addition, the umbilical cord is inflamed with multiple areas of abscess-like foci of necrosis surrounding the umbilical vessels. Which of the following is the most likely cause of these findings?

- ☐ A. ACE inhibitor exposure
- ☐ B. Partial hydatidiform mole
- ☐ C. Placenta accreta
- ☐ D. Rh(D) incompatibility
- ☐ E. Spirochete infection

Submit

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- A. ACE inhibitor exposure (11%)
- ✗

B. Partial hydatidiform mole (2%)
- C. Placenta accreta (8%)
- D. Rh(D) incompatibility (32%)
- ✓

E. Spirochete infection (44%)

Incorrect

Correct answer
E

44%
Answered correctly

22 secs
Time Spent

2023
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Explanation

Clinical features of early congenital syphilis	
Perinatal	<ul style="list-style-type: none">Intrauterine fetal demisePrematurity, low birth weightFocal necrosis of the umbilical cord

Clinical features of early congenital syphilis	
Perinatal	<ul style="list-style-type: none">• Intrauterine fetal demise• Prematurity, low birth weight• Focal necrosis of the umbilical cord
Mucocutaneous	<ul style="list-style-type: none">• Snuffles: copious rhinorrhea, often purulent or serosanguineous• Desquamating, maculopapular rash involving palms/soles• Fissures near orifices (eg, anal, oral)• Jaundice
Musculoskeletal	<ul style="list-style-type: none">• Long bone abnormalities (eg, periosteal thickening, metaphyseal erosion)
Reticuloendothelial	<ul style="list-style-type: none">• Hepatomegaly ± splenomegaly• Lymphadenopathy• Anemia, thrombocytopenia, leukopenia/leukocytosis

Patients with an **atypical delivery** (eg, <37 weeks gestation [preterm], small for gestational age) undergo placental evaluation to search for an underlying cause (eg, infection, placental insufficiency). In this patient, histopathology shows umbilical cord inflammation with **abscess-like foci of necrosis** surrounding the **umbilical vessels**. This constellation of findings (ie, umbilical cord necrosis, small for gestational age) suggests an infectious etiology, likely **congenital infection**.

In this patient, the most likely cause is congenital **syphilis**, which occurs when *Treponema pallidum* **spirochetes** in the maternal circulation attach to and traverse the endothelial cells of the umbilical vein, thereby spreading to the placenta and fetus (**transplacental transmission**). In response, fetal inflammatory cells are recruited along the umbilical vessels, causing cord inflammation and perivascular necrosis (necrotizing funisitis). With placental compromise, fetal perfusion decreases and leads to chronic fetal hypoxia and **growth restriction**. Because congenital syphilis can also cause severe fetal anemia, the placenta may appear grossly pale and enlarged with

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Newborns with suspected congenital syphilis require nontreponemal testing (eg, rapid reagin test, VDRL). Affected infants require penicillin therapy to prevent late manifestations (eg, [saddle nose](#), [notched \[Hutchinson\] teeth](#)). Routine prenatal care usually prevents congenital syphilis by screening for and treating maternal syphilis.

(Choice A) ACE inhibitors cross the placenta and are associated with fetal growth restriction as well as renal dysfunction, oligohydramnios, and neonatal respiratory distress. ACE inhibitor exposure does not cause umbilical cord inflammation or perivascular necrosis, which are signs of infection.

(Choice B) Partial hydatidiform moles, which typically result from fertilization of an ovum by 2 sperm, can cause enlarged, hydropic placental villi; the placenta typically has foci of trophoblastic proliferation.

(Choice C) [Placenta accreta spectrum](#) occurs when placental villi implant on or invade into the uterine myometrium. Placental examination typically reveals missing (retained) placental tissue and adherent myometrial fibers.

(Choice D) [Rh\(D\) incompatibility](#) which occurs when maternal anti-D antibodies attack Rh(D)-positive fetal red

In this patient, the most likely cause is congenital **syphilis**, which occurs when *Treponema pallidum* **spirochetes** in the maternal circulation attach to and traverse the endothelial cells of the umbilical vein, thereby spreading to the placenta and fetus (**transplacental transmission**). In response, fetal inflammatory cells are recruited along the umbilical vessels, causing cord inflammation and perivascular necrosis (necrotizing funisitis). With placental compromise, fetal perfusion decreases and leads to chronic fetal hypoxia and **growth restriction**. Because congenital syphilis can also cause severe fetal anemia, the placenta may appear grossly pale and enlarged with hydropic villi.

Newborns with suspected congenital syphilis require nontreponemal testing (eg, rapid reagin test, VDRL). Affected infants require penicillin therapy to prevent late manifestations (eg, [saddle nose](#), [notched \[Hutchinson\] teeth](#)). Routine prenatal care usually prevents congenital syphilis by screening for and treating maternal syphilis.

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(Choice B) Partial hydatidiform moles, which typically result from fertilization of an ovum by 2 sperm, can cause enlarged, hydropic placental villi; the placenta typically has foci of trophoblastic proliferation.

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(Choice D) [Rh\(D\) incompatibility](#), which occurs when maternal anti-D antibodies attack Rh(D)-positive fetal red blood cells, can lead to fetal anemia, growth restriction, and fetal and placental hydrops (eg, pale, enlarged placenta). However, it does not cause umbilical cord inflammation with abscess-like foci of necrosis.

Educational objective:

Patients with an atypical delivery (eg, preterm) require placental evaluation. Umbilical cord inflammation with abscess-like foci of necrosis suggests congenital syphilis (ie, spirochete infection with *Treponema pallidum*).

- Hepatomegaly ± splenomegaly

Exhibit Display

Saddle nose & perioral fissures



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(Choice C) Placenta accreta spectrum occurs when placental villi implant on or invade into the uterine

- Hepatomegaly ± splenomegaly

Exhibit Display

Hutchinson teeth



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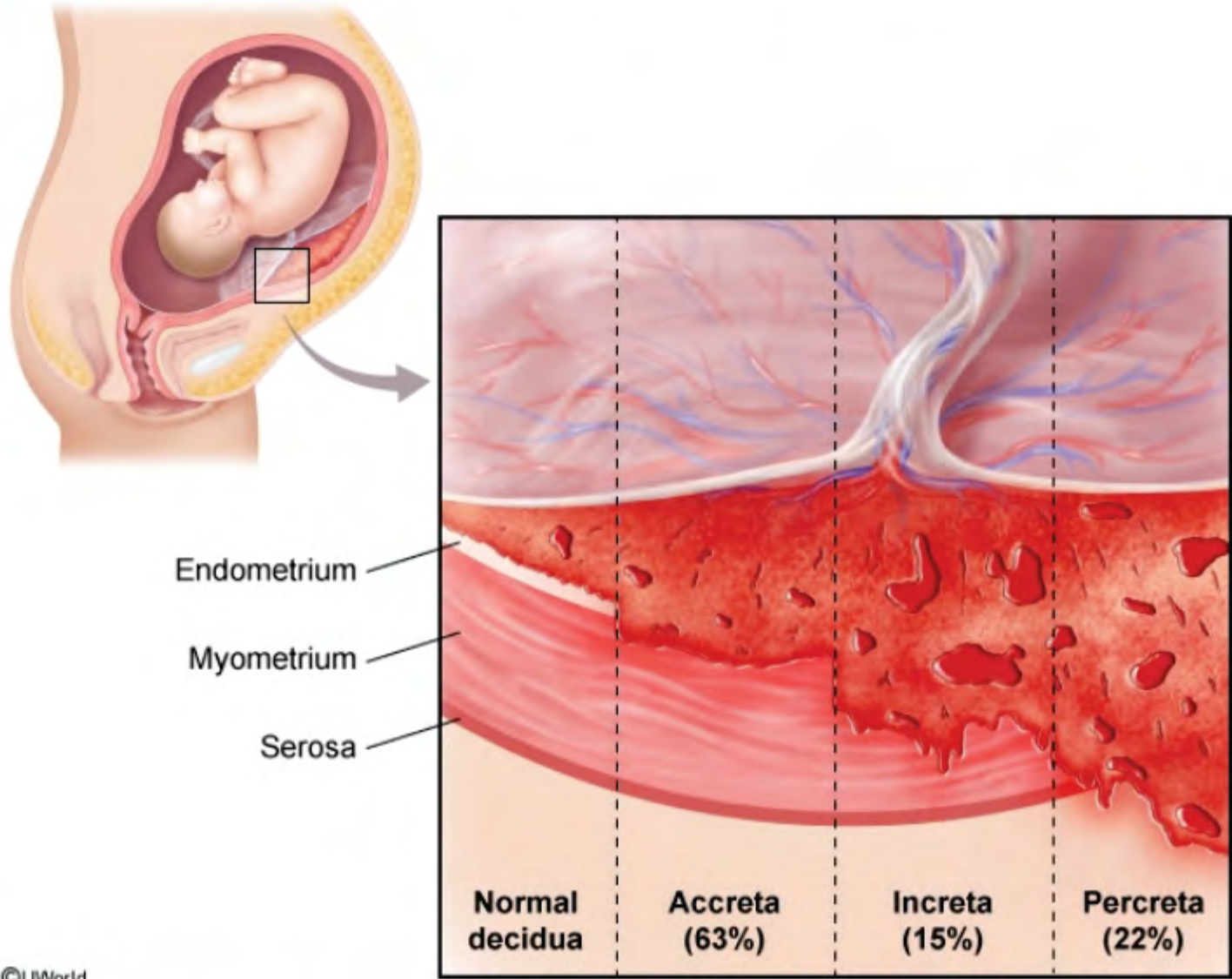
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(Choice C) Placenta accreta spectrum occurs when placental villi implant on or invade into the uterine

In this patient, the most likely cause is congenital **syphilis**, which occurs when *Treponema pallidum* **spirochetes** in the maternal circulation attach to and traverse the endothelial cells of the umbilical vein, thereby spreading to the placenta.

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Placenta accreta



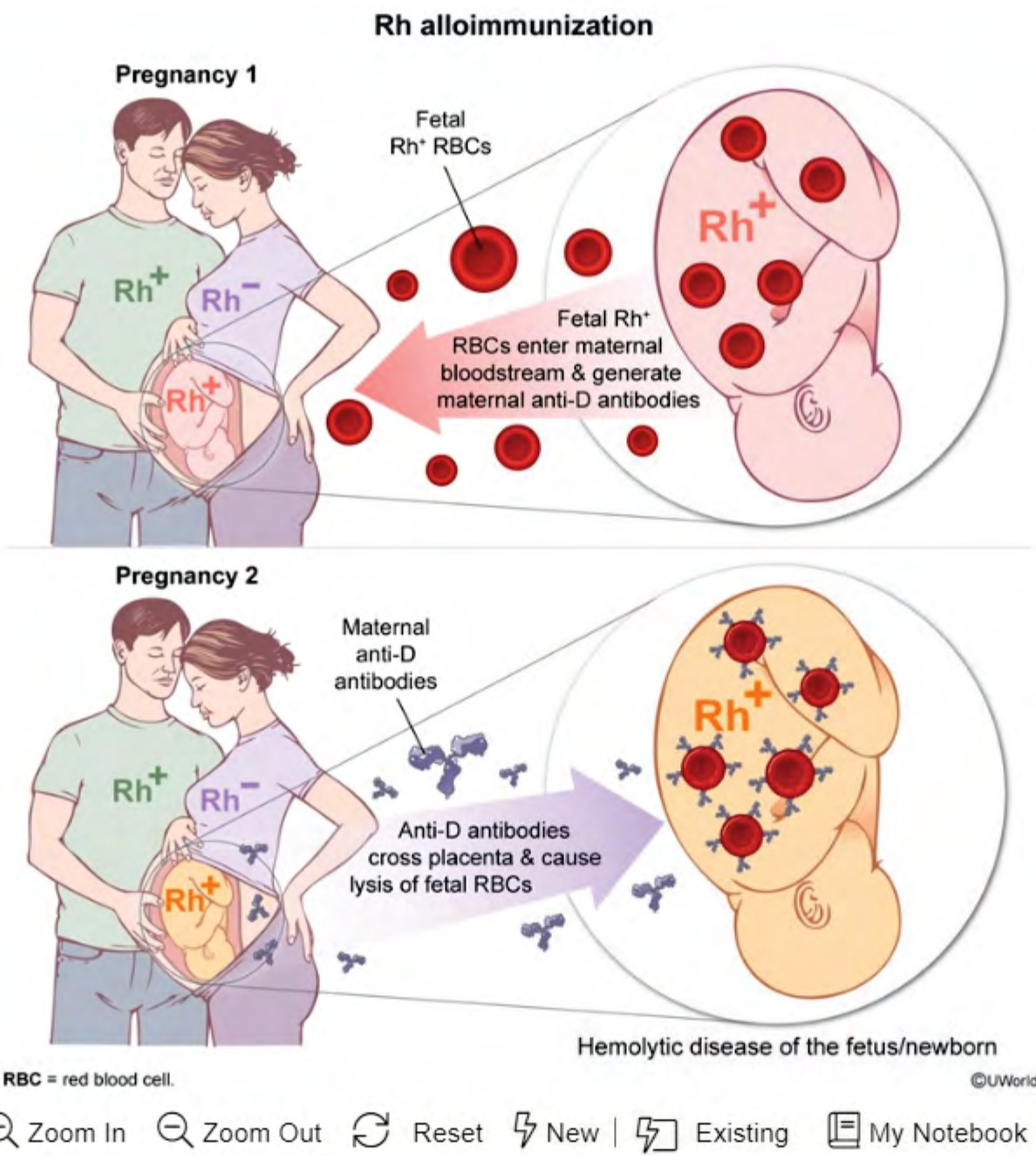
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abscess-like foci of necrosis suggests congenital syphilis (ie, spirochete infection with *Treponema pallidum*).

In this patient, the most likely cause is congenital **syphilis**, which occurs when *Treponema pallidum* **spirochetes** in the maternal circulation attach to and traverse the endothelial cells of the umbilical vein, thereby spreading to the placenta.

Exhibit Display



abscess-like foci of necrosis suggests congenital syphilis (ie, spirochete infection with *Treponema pallidum*).

A 25-year-old woman comes to the office due to amenorrhea. The patient's last menstrual period was 8 weeks ago, and she normally has regular, monthly menses. The patient has no chronic medical conditions and has had no surgeries. She takes no daily medications. Blood pressure is 100/60 mm Hg and pulse is 92/min. BMI is 24 kg/m². Abdominal examination shows a soft, nontender abdomen with no palpable masses. Pelvic examination shows an 8-week-sized, nontender uterus. Serum β -hCG level is elevated. Which of the following is the most important direct role of hCG in this patient?

- ☐ A. Induction of prolactin production by the pituitary
- ☐ B. Inhibition of uterine contractions
- ☐ C. Initiation of embryonic cell division and differentiation
- ☐ D. Maintenance of the corpus luteum
- ☐ E. Promotion and maintenance of embryonic implantation

Submit

A 25-year-old woman comes to the office due to amenorrhea. The patient's last menstrual period was 8 weeks ago, and she normally has regular, monthly menses. The patient has no chronic medical conditions and has had no surgeries. She takes no daily medications. Blood pressure is 100/60 mm Hg and pulse is 92/min. BMI is 24 kg/m². Abdominal examination shows a soft, nontender abdomen with no palpable masses. Pelvic examination shows an 8-week-sized, nontender uterus. Serum β -hCG level is elevated. Which of the following is the most important direct role of hCG in this patient?

- A. Induction of prolactin production by the pituitary (0%)

✖

B. Inhibition of uterine contractions (1%)

C. Initiation of embryonic cell division and differentiation (3%)

✔

D. Maintenance of the corpus luteum (79%)

E. Promotion and maintenance of embryonic implantation (15%)

Incorrect

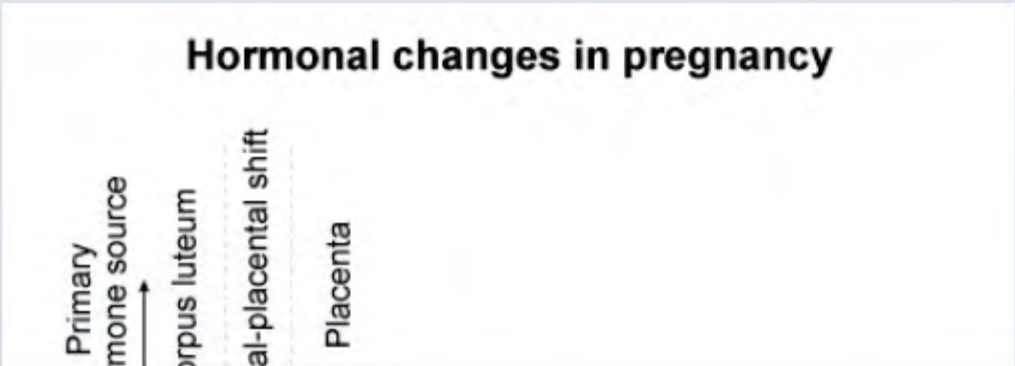
Correct answer
D

79%
Answered correctly

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Time Spent

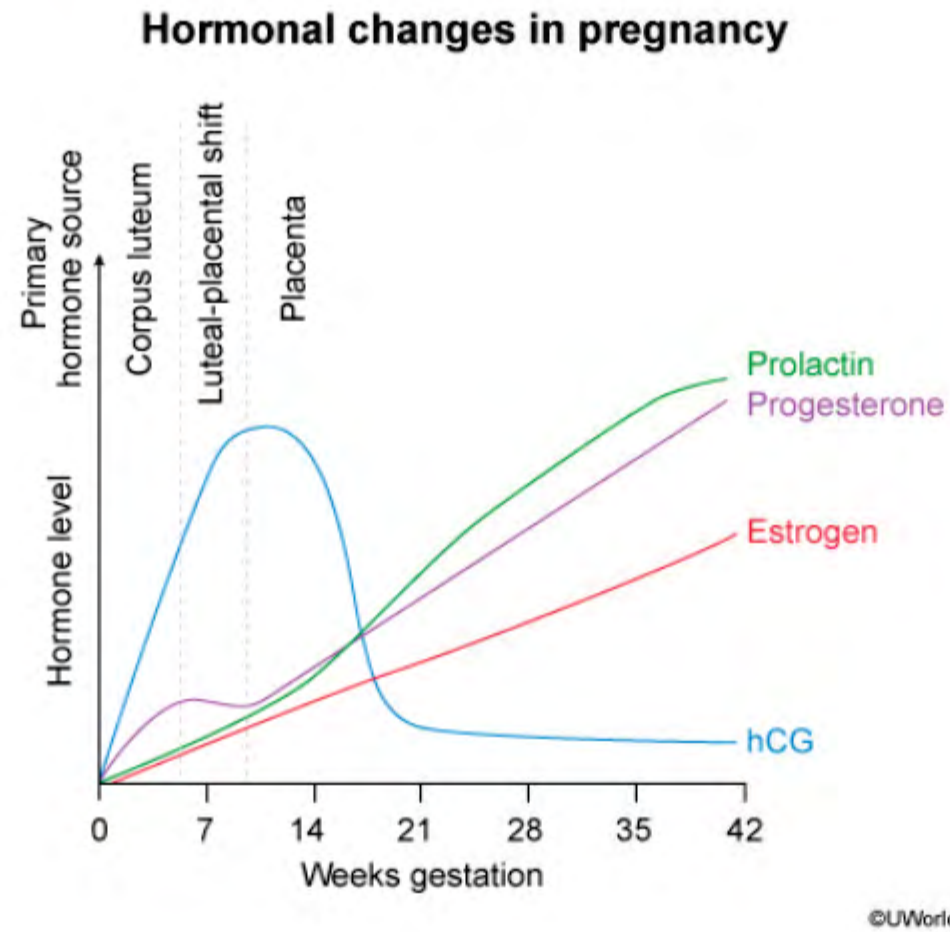
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Explanation



B. Inhibition of uterine contractions (1%)

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Weeks gestation

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Early pregnancy depends on hormonal feedback between the ovary, uterine endometrium, and embryo. In a normal [menstrual cycle](#), ovulation (ie, oocyte release) creates the **ovarian corpus luteum**, which increases **progesterone production** to induce endometrial decidualization (ie, uterine lining thickening, formation of the decidua basalis) in preparation for both promotion and maintenance of embryonic implantation (**Choice E**).

When the oocyte is fertilized, a blastocyst eventually develops and implants into the uterine decidua basalis approximately 6-7 days after fertilization. The outer cells of the blastocyst (ie, trophoblast) then differentiate into the [cytotrophoblast and syncytiotrophoblast](#), which develop into the placenta.

The **syncytiotrophoblast** invades the decidua basalis and **secretes β -hCG** into maternal circulation. β -hCG signals the ovary to **maintain the corpus luteum** and to continue progesterone production, which maintains both the decidualized endometrium and the pregnancy until the placenta has developed enough to completely take over progesterone production (typically by 10 weeks gestation). Following this **luteal-placental transition**, the corpus luteum degenerates, and the role of progesterone changes to maintain uterine quiescence (ie, inhibit contractions) (**Choice B**).

(**Choice A**) Estrogen, not hCG, stimulates the anterior pituitary to produce prolactin during pregnancy.

(**Choice C**) The process of [embryonic cell division](#) and differentiation begins before the formation of the placental syncytiotrophoblast, which secretes hCG; therefore, hCG does not cause embryonic cell division.

Educational objective:

β -hCG is a hormone secreted by the placental syncytiotrophoblast after uterine invasion that signals the ovary to maintain the corpus luteum, which produces progesterone in early pregnancy.

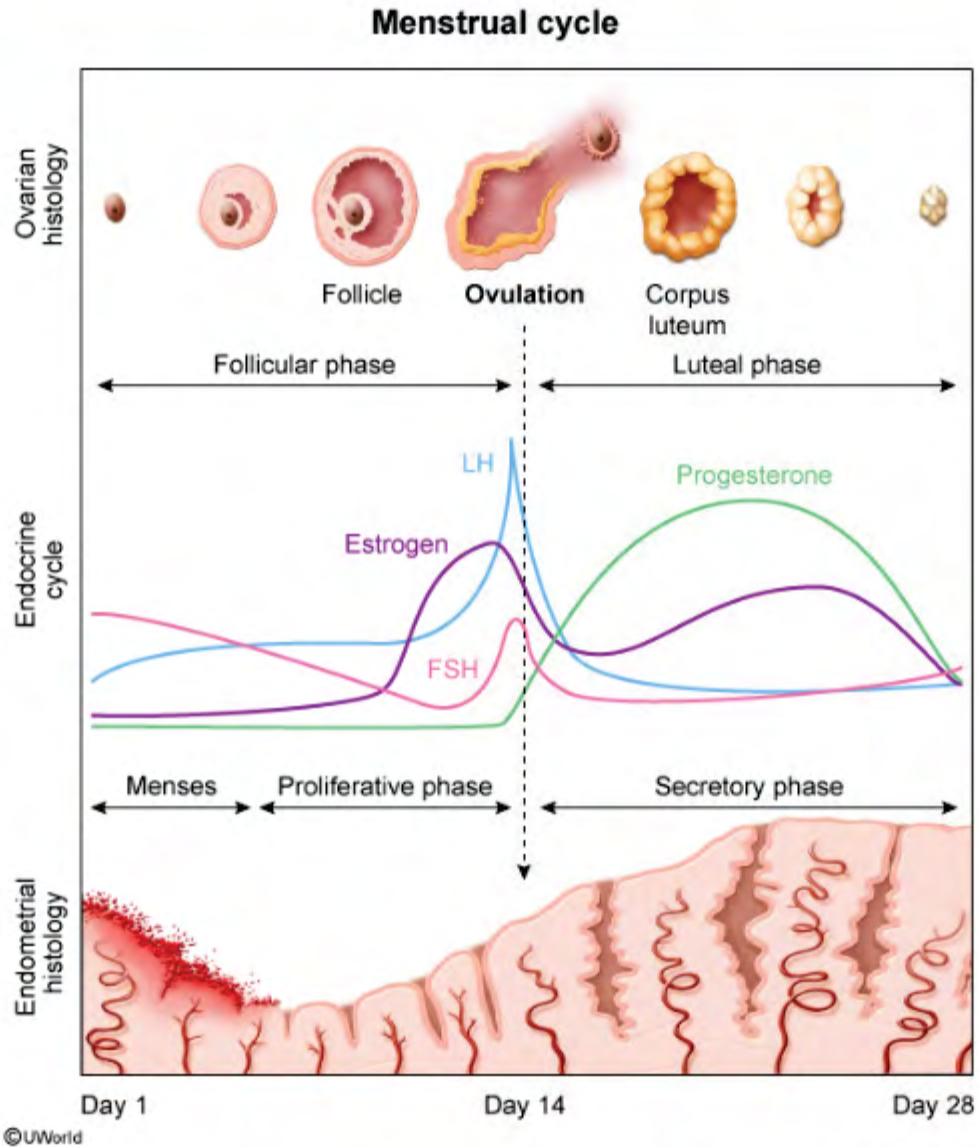
References

- [hCG: biological functions and clinical applications.](#)



Weeks gestation

Exhibit Display



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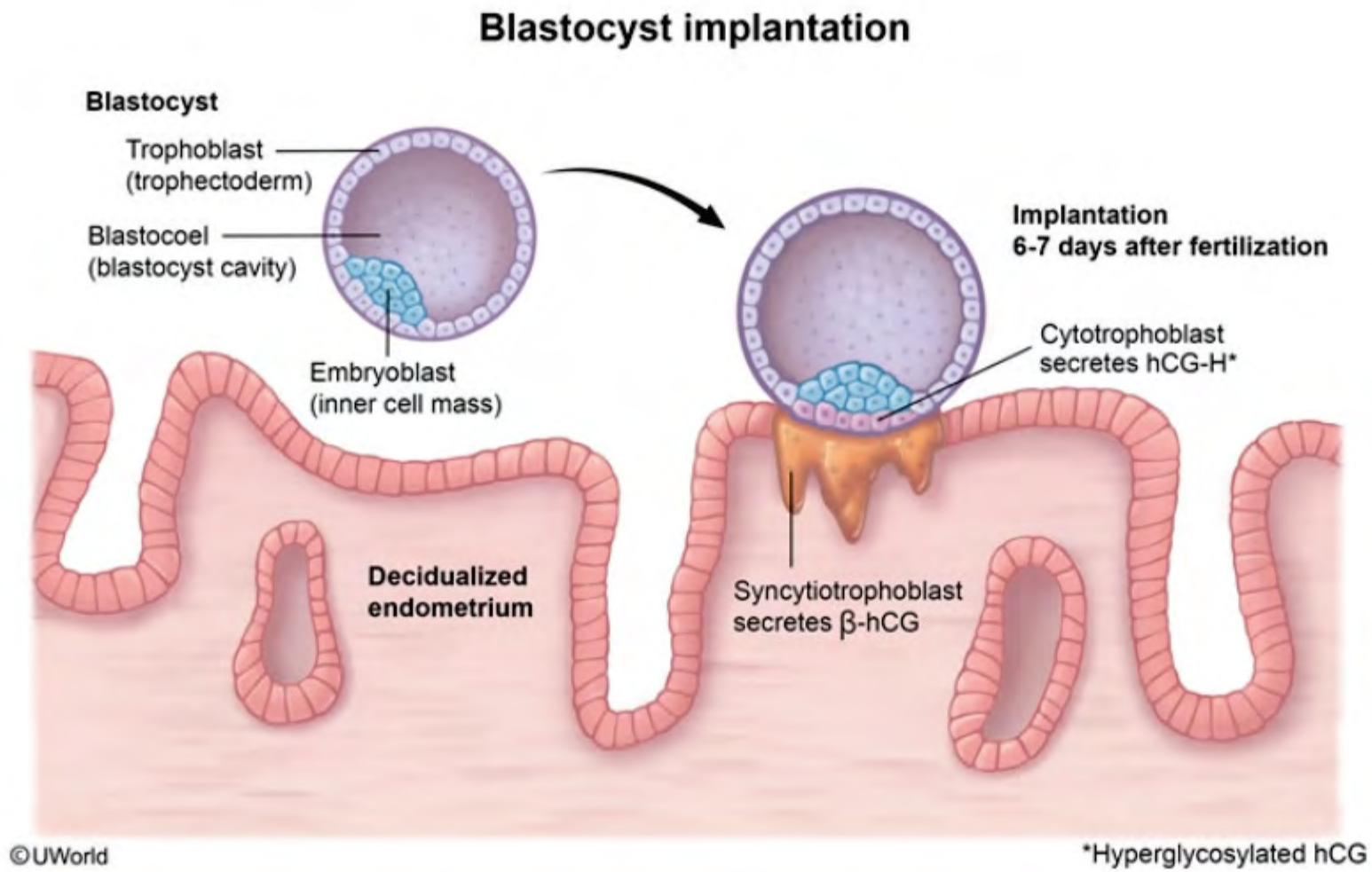
References

- hCG: biological functions and clinical applications.

Weeks gestation

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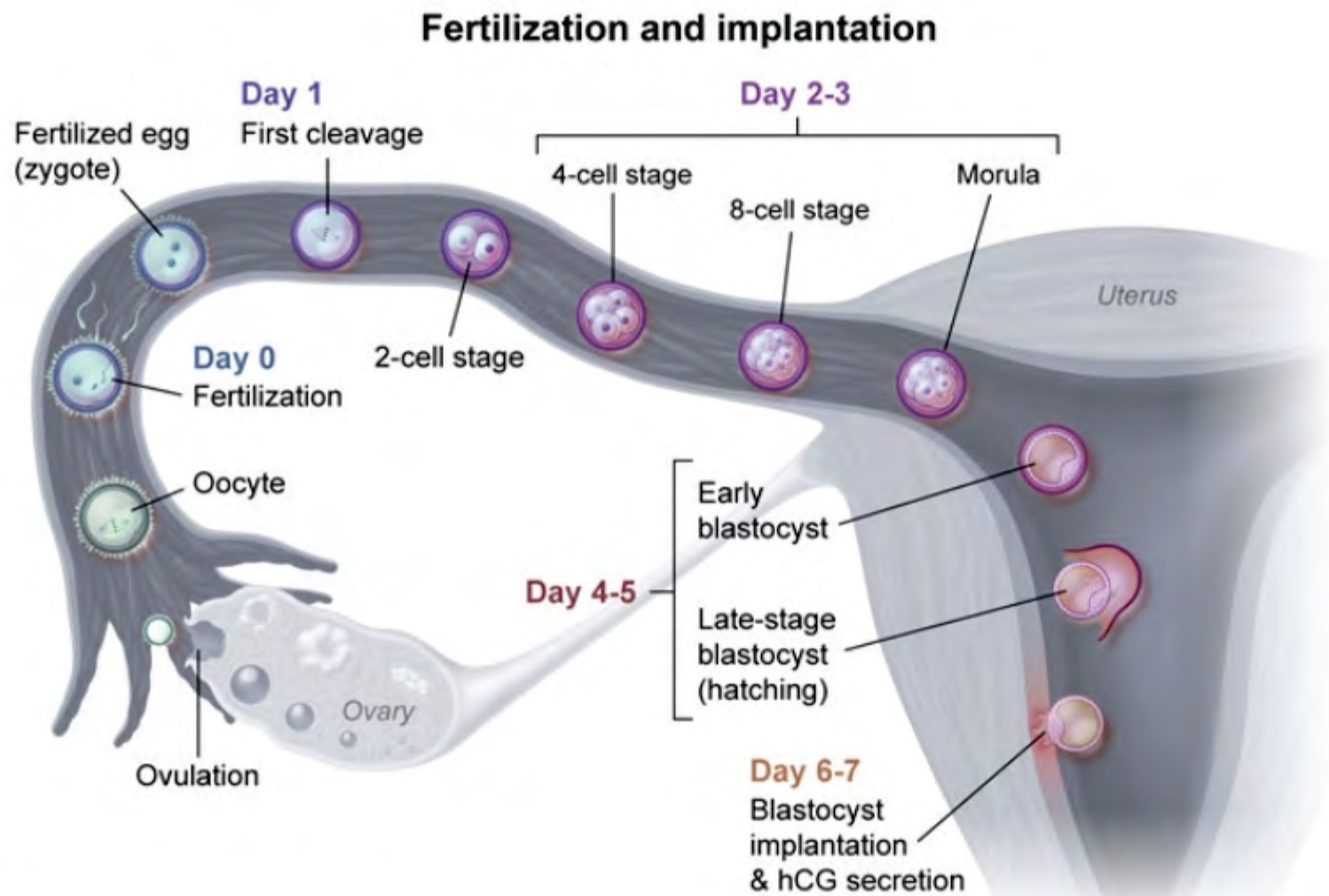


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Weeks gestation

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A 32-year-old woman, gravida 2 para 1, has a spontaneous vaginal delivery. Immediately after delivery of the placenta, the patient has a large amount of vaginal bleeding with passage of fist-sized clots, findings consistent with postpartum hemorrhage. A dose of tranexamic acid is administered. Which of the following is the most likely mechanism of action of this drug?

- ☐ A. Increases vitamin K–dependent coagulation factor levels
- ☐ B. Induces prostaglandin-mediated smooth muscle contraction
- ☐ C. Inhibits plasminogen cleavage and prevents fibrin degradation
- ☐ D. Promotes endothelial release of von Willebrand factor
- ☐ E. Raises intracellular calcium levels in the uterine myometrium

Submit

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- A. Increases vitamin K–dependent coagulation factor levels (8%)

✖

B. Induces prostaglandin-mediated smooth muscle contraction (14%)

✔

C. Inhibits plasminogen cleavage and prevents fibrin degradation (66%)

D. Promotes endothelial release of von Willebrand factor (4%)

E. Raises intracellular calcium levels in the uterine myometrium (5%)

Incorrect

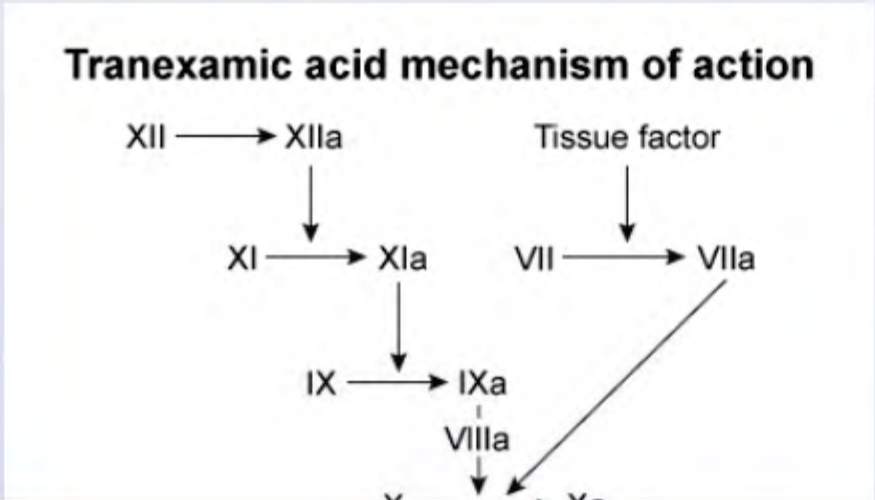
Correct answer
C

66%
Answered correctly

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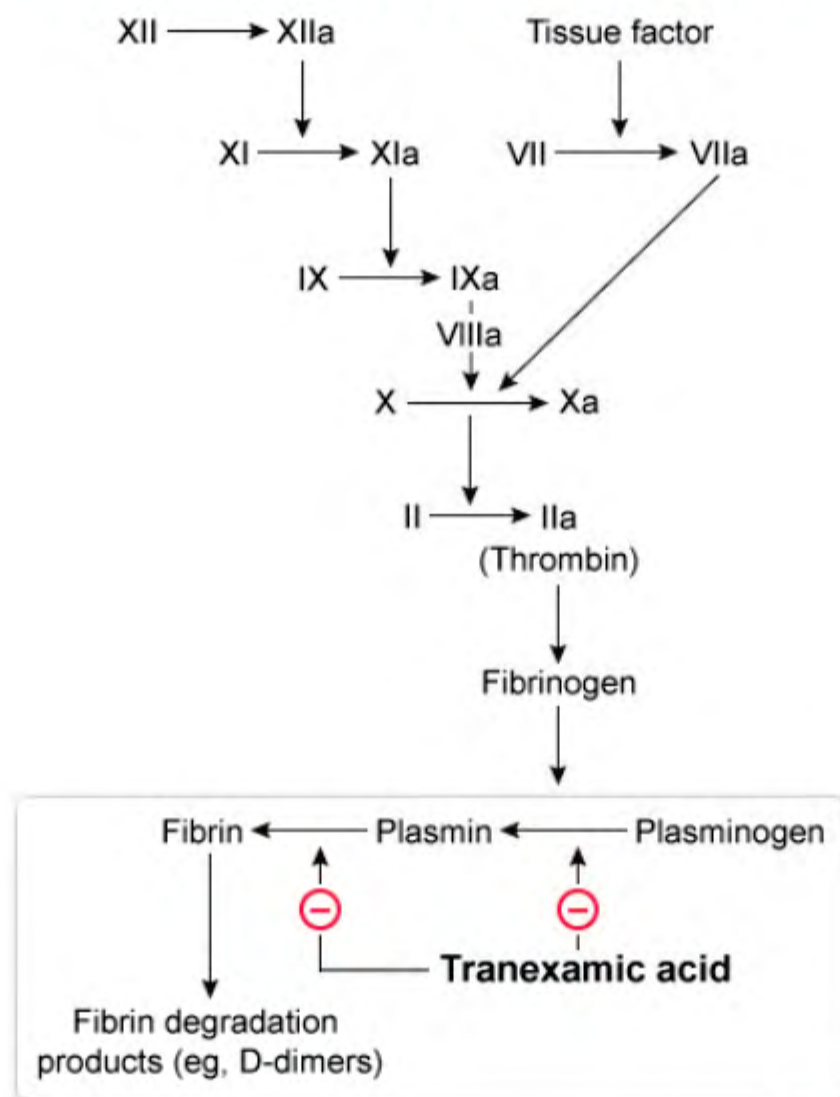
Explanation



☐ D. Promotes endothelial release of von Willebrand factor (4%)

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Tranexamic acid mechanism of action



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This patient has **postpartum hemorrhage** (PPH). Following placental delivery, maternal **hemostasis** is typically achieved by **uterine contraction**, which constrict bleeding placental bed vessels, and activation of coagulation pathways and **clot formation** at the former placental attachment site. When either mechanism fails, patients may have PPH and life-threatening blood loss. Therefore, medications used to treat PPH commonly increase either uterine contractions or clot formation.

After placental delivery, prothrombotic substances in the decidua (eg, tissue factor) activate the extrinsic coagulation cascade, generating thrombin that converts fibrinogen into fibrin, forming a fibrin clot. At the same time, the **fibrinolytic pathway** is also activated: tissue plasminogen activator (t-PA) is released by damaged endothelium and cleaves plasminogen to form plasmin, which promotes bleeding by breaking down fibrin clots into fibrin degradation products.

Tranexamic acid (TXA) stabilizes thrombi/clots by **inhibiting plasmin formation**; therefore, TXA is an **antifibrinolytic medication** that provides more time for the uterine myometrium to contract and constrict the placental bed blood vessels. The use of TXA for PPH significantly decreases bleeding and is associated with improved maternal morbidity and mortality.

(Choice A) Increasing vitamin K–dependent coagulation factor levels is the mechanism of action for prothrombin complex concentrate (PCC), which contains factors II, IX, and X and variable amounts of factor VII. PCC is commonly used to reverse warfarin-induced anticoagulation or to treat bleeding due to congenital vitamin K–dependent coagulation factor deficiencies.

(Choices B and E) Agents that cause uterine myometrial contractions are known as uterotonic medications. Misoprostol, a synthetic prostaglandin analogue, induces prostaglandin-mediated smooth muscle contraction but has a slow onset of action. In contrast, oxytocin rapidly increases intracellular calcium levels in the uterine myometrium; because of its rapid onset, is the most common uterotonic administered for PPH.

(Choice D) Promotion of the endothelial release of von Willebrand factor is the mechanism of action for desmopressin, which also increases plasma levels of factor VIII. Desmopressin is used in patients with bleeding

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(Choice D) Promotion of the endothelial release of von Willebrand factor is the mechanism of action for desmopressin, which also increases plasma levels of factor VIII. Desmopressin is used in patients with bleeding due to von Willebrand disease or mild hemophilia A.

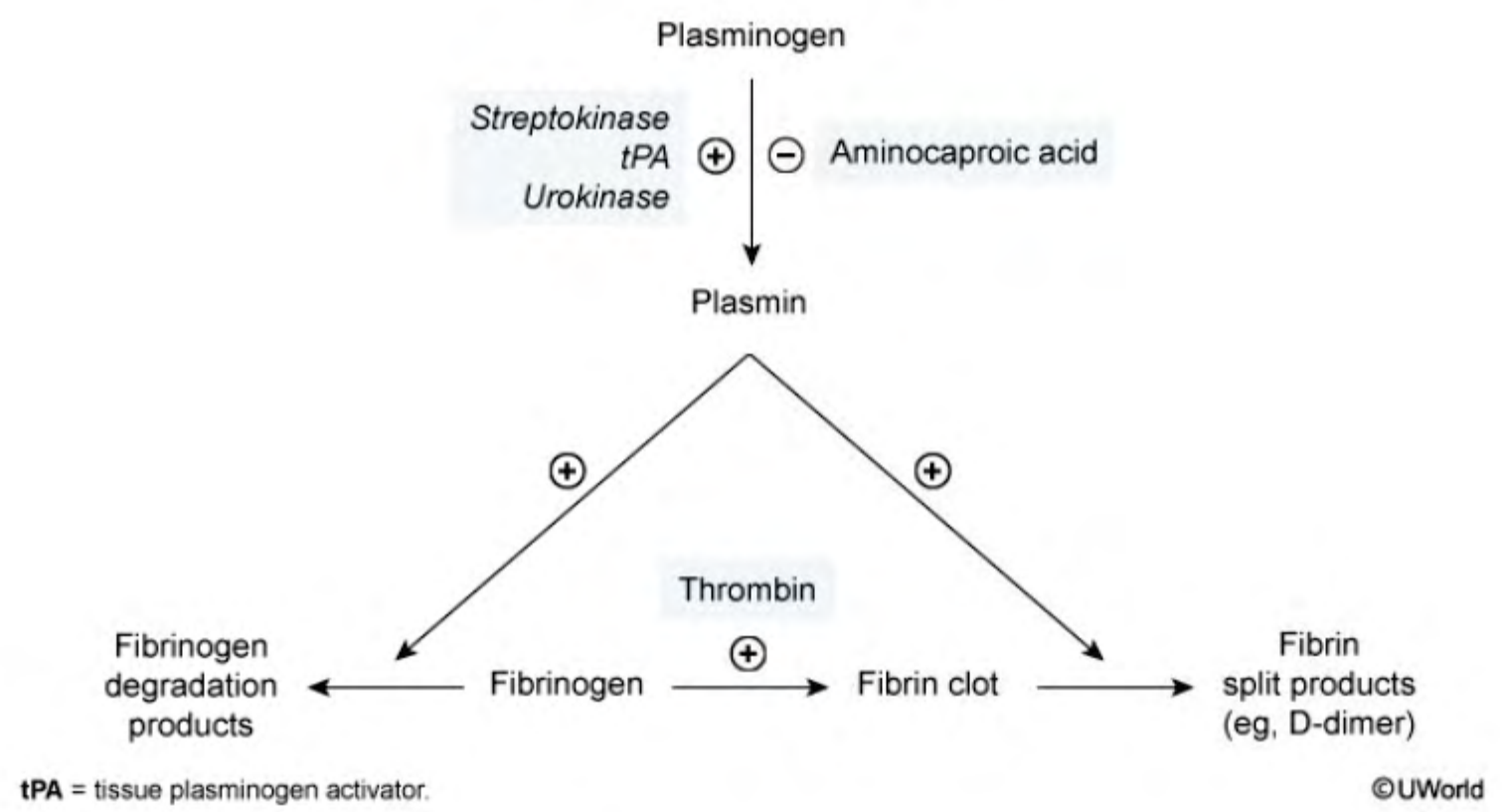
Educational objective:

Tranexamic acid (TXA) is an antifibrinolytic medication that stabilizes blood clots in hemorrhaging patients. It inhibits plasmin formation, thereby preventing fibrin degradation and clot dissolution. TXA used for the treatment of postpartum hemorrhage improves maternal morbidity and mortality.

Tranexamic acid

Exhibit Display

Fibrinolytic pathway



A 31-year-old woman, gravida 3 para 2, at 28 weeks gestation comes to the office for a prenatal visit. She has chronic hypertension that has been well controlled during this pregnancy. At the patient's previous prenatal visits, her blood pressure ranged from 120/70 to 130/80 mm Hg. Today, her blood pressure is 150/100 mm Hg and repeat is 152/98 mm Hg. Fundal height is 24 cm. Urinalysis shows 2+ protein. A transabdominal ultrasound reveals oligohydramnios and a fetus with growth restriction. Compared to normal placental parameters, this patient most likely has which of the following hemodynamic changes?

	Placental vascular resistance	Uteroplacental perfusion	Umbilical vein oxygen delivery
<input type="radio"/> A.	↓	↓	↑
<input type="radio"/> B.	↓	↑	↑
<input type="radio"/> C.	No change	↓	↓
<input type="radio"/> D.	↑	↓	No change
<input type="radio"/> E.	↑	↓	↑
<input type="radio"/> F.	↑	↓	↓

Submit

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	Placental vascular resistance	Uteroplacental perfusion	Umbilical vein oxygen delivery	
<input type="radio"/> A.	↓	↓	↑	(1%)
<input checked="" type="radio"/> B.	↓	↑	↑	(1%)
<input type="radio"/> C.	No change	↓	↓	(4%)
<input type="radio"/> D.	↑	↓	No change	(16%)
<input type="radio"/> E.	↑	↓	↑	(2%)
<input checked="" type="radio"/> F.	↑	↓	↓	(72%)

Incorrect

Correct answer F

72% Answered correctly

03 secs Time Spent

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Explanation

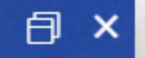
Pathogenesis of preeclampsia

Incorrect

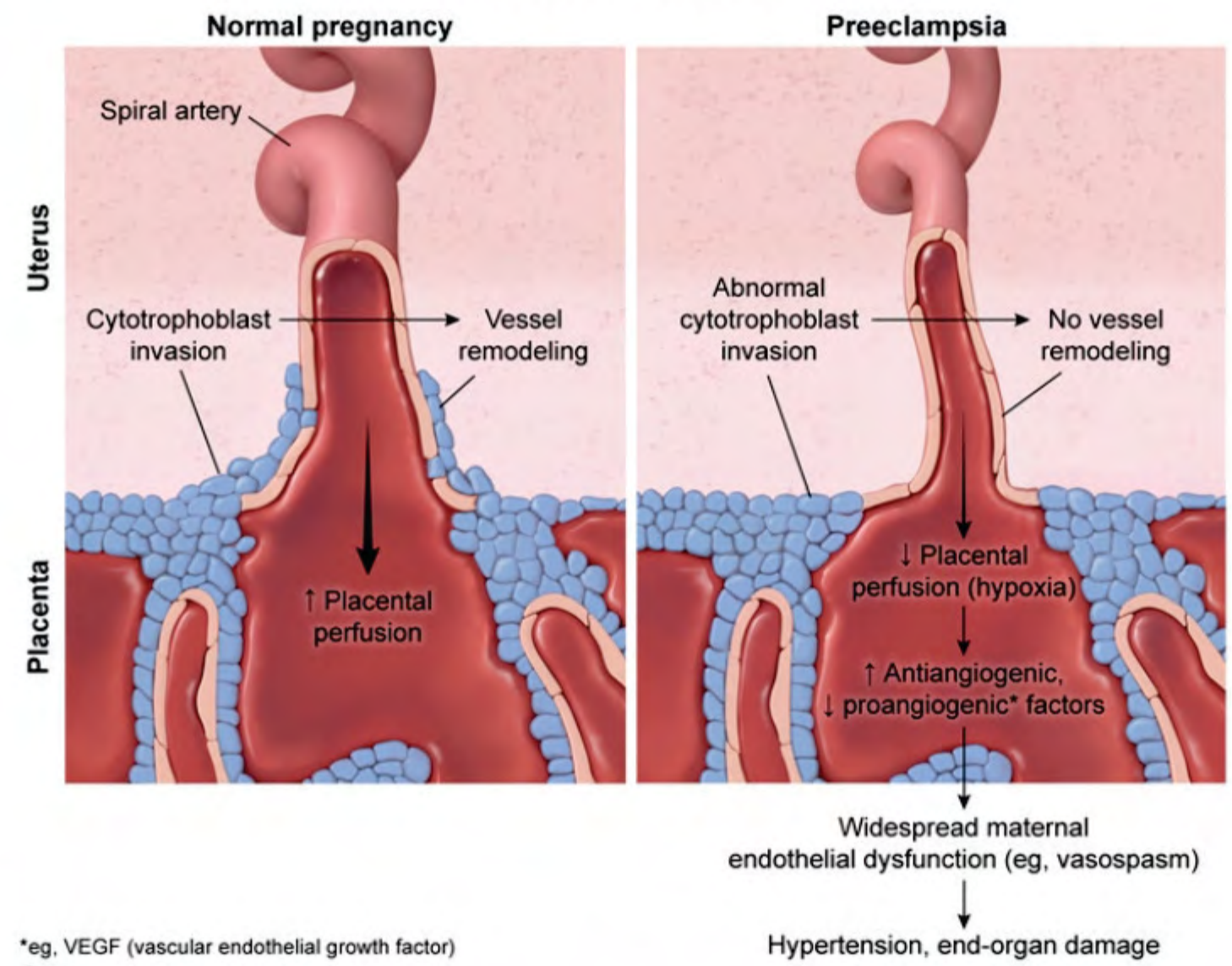
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Pathogenesis of preeclampsia



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*eg, VEGF (vascular endothelial growth factor)

Hypertension, end-organ damage

<https://t.me/USMLEWorldStep1>

*eg, VEGF (vascular endothelial growth factor)
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Hypertension, end-organ damage

This pregnant patient at ≥ 20 weeks gestation with hypertension (defined during pregnancy as systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg) and proteinuria has **preeclampsia**. Risk factors include chronic hypertension or maternal vascular diseases (eg, diabetes mellitus, systemic lupus erythematosus).

Preeclampsia is thought to occur due to abnormal development of placental cytotrophoblasts and the spiral arteries, which provide blood supply to the fetus and placenta. In healthy pregnancies, cytotrophoblasts act on the endothelial and muscular layers of the spiral arteries, changing the spiral arteries from small, tortuous vessels to high capacitance, low resistance vessels. These changes allow for increased uteroplacental perfusion and preferential supply of blood to the fetus for growth and development.

However, in patients with preeclampsia, spiral arteries remain narrow and tortuous, resulting in **high placental vascular resistance**. This subsequently leads to **decreased uteroplacental perfusion**. As a result, the placenta cannot deliver adequate volumes of oxygenated blood to the fetus (ie, **decreased umbilical vein oxygen delivery**). The fetal response to inadequate oxygenation is to slow its growth (ie, **fetal growth restriction**) and preferentially shunt blood from the periphery (eg, kidneys) to the brain, leading to decreased fetal urine output and **oligohydramnios**, as seen in this patient.

Educational objective:

Patients with preeclampsia are at risk for fetal growth restriction and oligohydramnios due to abnormal placental spiral artery development, which leads to increased placental vascular resistance, decreased uteroplacental perfusion, and decreased umbilical vein oxygen delivery.

References

- [Mechanisms of endothelial dysfunction in hypertensive pregnancy and preeclampsia.](#)

A 25-year-old primigravida at 37 weeks gestation is brought to the emergency department with constant, excruciating abdominal pain and sudden vaginal bleeding for the past 3 hours. The patient is Rh negative and received Rh(D) immunoglobulin at 28 weeks gestation. She has no chronic medical conditions and takes no medications. Blood pressure is 160/100 mm Hg and pulse is 118/min. Physical examination shows a firm and tender uterus. Speculum examination reveals a moderate amount of bleeding coming from an open cervical os. Fetal heart tracing shows a baseline of 105/min and no variability. Which of the following is the most likely cause of this patient's bleeding?

- ☐

A. Attachment of the placenta onto the myometrium
- ☐

B. Full-thickness disruption of the uterine wall
- ☐

C. Injury to the fetal placental vessels
- ☐

D. Placental implantation over the cervix
- ☐

E. Premature placental separation

Submit

A 25-year-old primigravida at 37 weeks gestation is brought to the emergency department with constant, excruciating abdominal pain and sudden vaginal bleeding for the past 3 hours. The patient is Rh negative and received Rh(D) immunoglobulin at 28 weeks gestation. She has no chronic medical conditions and takes no medications. Blood pressure is 160/100 mm Hg and pulse is 118/min. Physical examination shows a firm and tender uterus. Speculum examination reveals a moderate amount of bleeding coming from an open cervical os. Fetal heart tracing shows a baseline of 105/min and no variability. Which of the following is the most likely cause of this patient's bleeding?

- A. Attachment of the placenta onto the myometrium (6%)
- ✗

B. Full-thickness disruption of the uterine wall (3%)
- C. Injury to the fetal placental vessels (6%)
- D. Placental implantation over the cervix (16%)
- ✓

E. Premature placental separation (66%)

Incorrect

Correct answer
E

66%
Answered correctly

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Explanation

Abruptio placentae	
Definition	<ul style="list-style-type: none">Premature placental separation from uterus
Risk factors	<ul style="list-style-type: none">Hypertension, preeclampsia

Abruptio placentae	
Definition	<ul style="list-style-type: none">• Premature placental separation from uterus
Risk factors	<ul style="list-style-type: none">• Hypertension, preeclampsia• Abdominal trauma• Cocaine or tobacco use• Prior abruptio placentae
Clinical features	<ul style="list-style-type: none">• Sudden-onset vaginal bleeding• Abdominal pain• High-frequency contractions• Tender, firm uterus

This patient in the third trimester with **painful vaginal bleeding** and a **tender, firm uterus** has a presentation consistent with **abruptio placentae** (ie, premature separation of the placenta from the uterus prior to fetal delivery). **Abruptio placentae** is associated with **maternal hypertension**, abdominal trauma, use of tobacco or cocaine (vasoconstrictors causing placental ischemia), and prior abruptio placentae.

Abruptio placentae occurs when maternal **vessels rupture at the uteroplacental interface**, causing intrauterine bleeding. This bleeding results in **detachment of the placenta** from the uterus, an increase in intrauterine pressure (eg, tender, distended uterus), and **uterine irritability** (eg, high-frequency, low-intensity contractions).

Some cases are self-limiting and contained, whereas others progress as the bleeding and placental separation continue. As the degree of placental separation increases, the risk of **fetal compromise** and mortality from hypoxia (eg, fetal bradycardia, minimal variability in the fetal heart rate tracing) also increases. Maternal complications include hemorrhage and **disseminated intravascular coagulation**. Acute abruptio placentae with active bleeding is an indication for delivery; the mode of delivery depends on both maternal and fetal stability.

(Choice A) **Placenta accreta**, direct attachment of the placental villi onto the myometrium, typically presents after

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(Choice A) **Placenta accreta**, direct attachment of the placental villi onto the myometrium, typically presents after delivery of the fetus with postpartum hemorrhage and inability to remove the placenta.

(Choice B) **Uterine rupture**, full-thickness disruption of the uterine wall, is associated with severe lower abdominal pain, vaginal bleeding, and fetal heart rate tracing abnormalities (eg, bradycardia). However, it typically occurs in patients with prior cesarean delivery (this patient is a primigravida) and typically has a uterus with palpable fetal parts, rather than uterine rigidity.

(Choice C) In **vasa previa**, the fetal vessels traverse the internal cervical os and are vulnerable to injury. However, patients typically have painless, rather than painful, vaginal bleeding.

(Choice D) **Placenta previa**, placental implantation over the cervix, typically presents in the third trimester with painless, rather than painful, vaginal bleeding and no uterine tenderness.

Educational objective:

cocaine (vasoconstrictors causing placental ischemia), and prior abruptio placentae.

Abruptio placentae occurs when maternal **vessels rupture at the uteroplacental interface**, causing intrauterine bleeding. This bleeding results in **detachment of the placenta** from the uterus, an increase in intrauterine pressure (eg, tender, distended uterus), and **uterine irritability** (eg, high-frequency, low-intensity contractions).

Some cases are self-limiting and contained, whereas others progress as the bleeding and placental separation continue. As the degree of placental separation increases, the risk of **fetal compromise** and mortality from hypoxia (eg, fetal bradycardia, minimal variability in the fetal heart rate tracing) also increases. Maternal complications include hemorrhage and **disseminated intravascular coagulation**. Acute abruptio placentae with active bleeding is an indication for delivery; the mode of delivery depends on both maternal and fetal stability.

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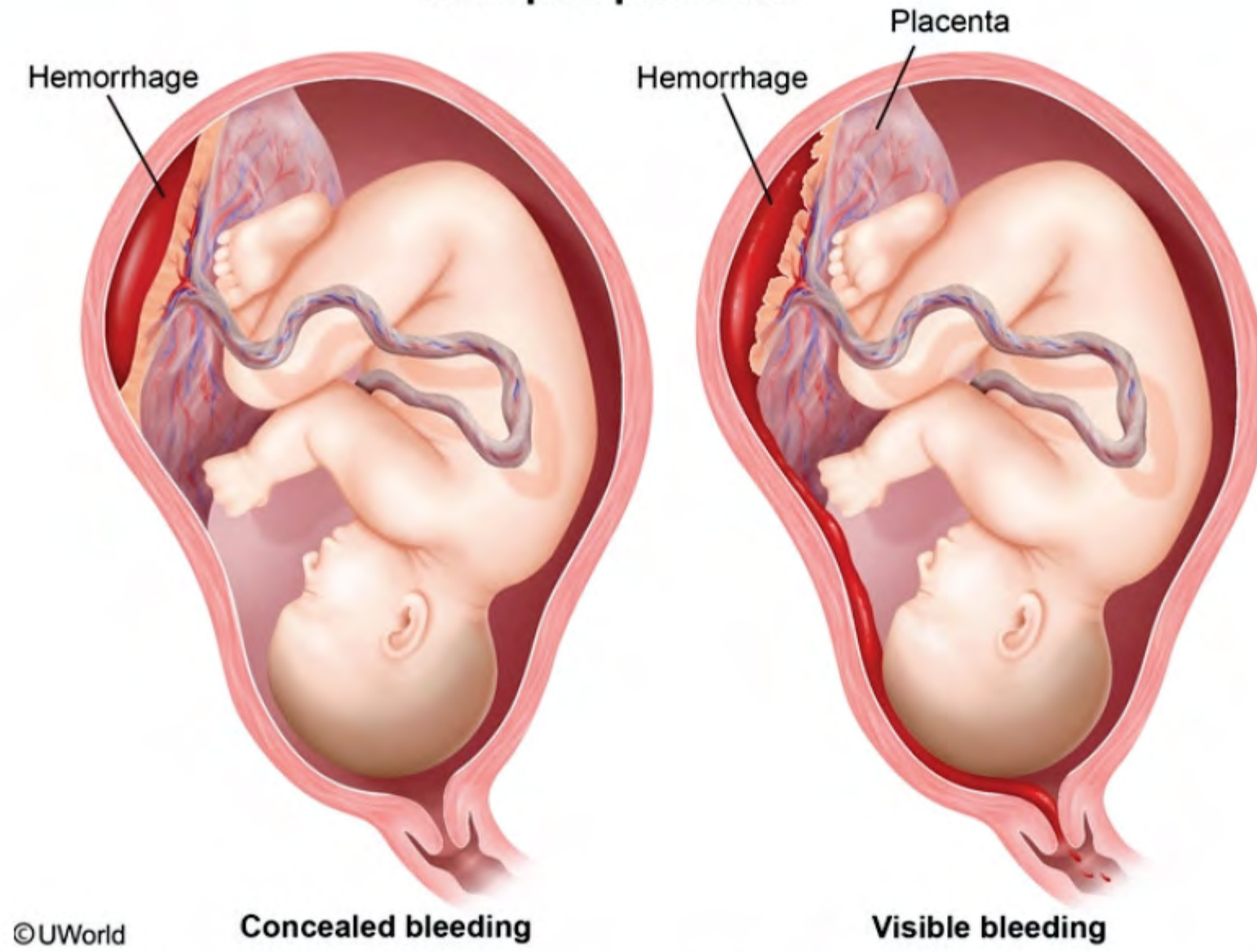
Educational objective:

Abruptio placentae, detachment of the placenta from the uterus prior to fetal delivery, presents with painful vaginal bleeding; a tender, firm uterus; and fetal heart rate abnormalities. Risk factors include abdominal trauma, maternal hypertension, and tobacco or cocaine use.

- Prior abruptio placentae

Exhibit Display

Abruptio placentae

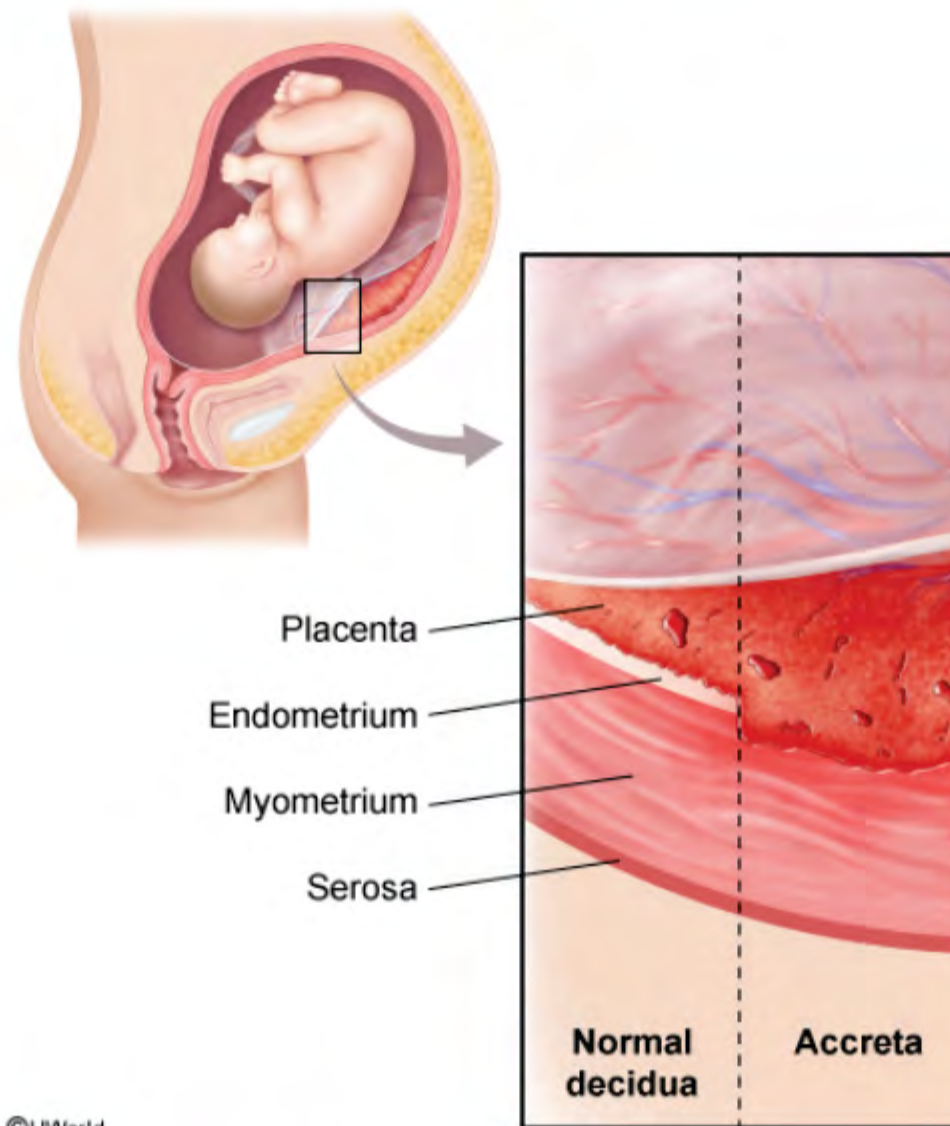


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- Prior abruptio placentae

Exhibit Display

Placenta accreta



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This patient in the third trimester with **painful vaginal bleeding** and a **tender, firm uterus** has a presentation

consistent with a placental abnormality. The patient is currently at fetal station 0.

delivered

cocaine

Abruptio

placental

presentation

Some

cont

hypertension

complications

active

(Choice)

delivered

(Choice)

painful

patient

parts

(Choice)

patient

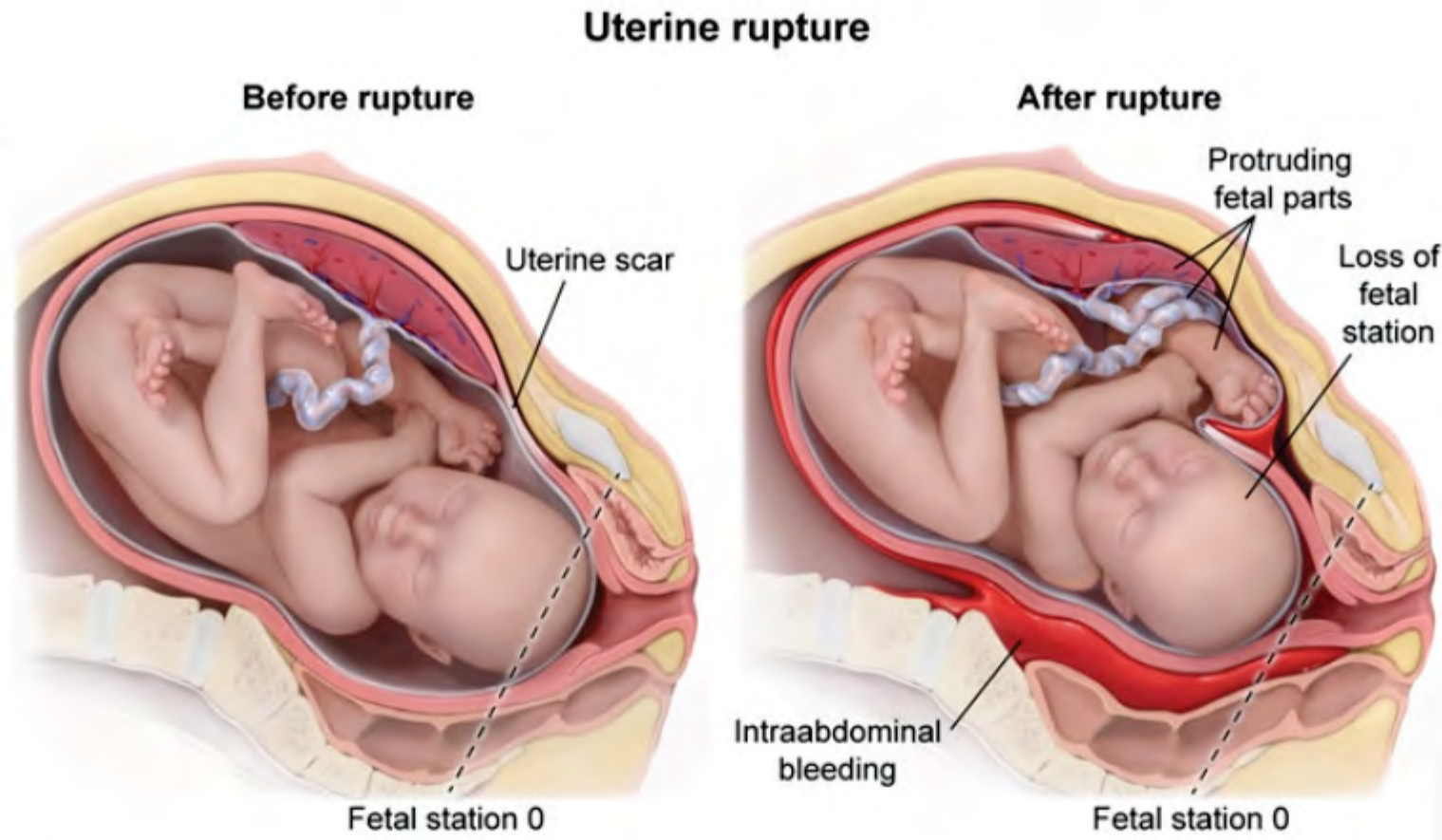
(Choice)

painful

Educational objective:

Abruptio placentae, detachment of the placenta from the uterus prior to fetal delivery, presents with painful vaginal

Exhibit Display



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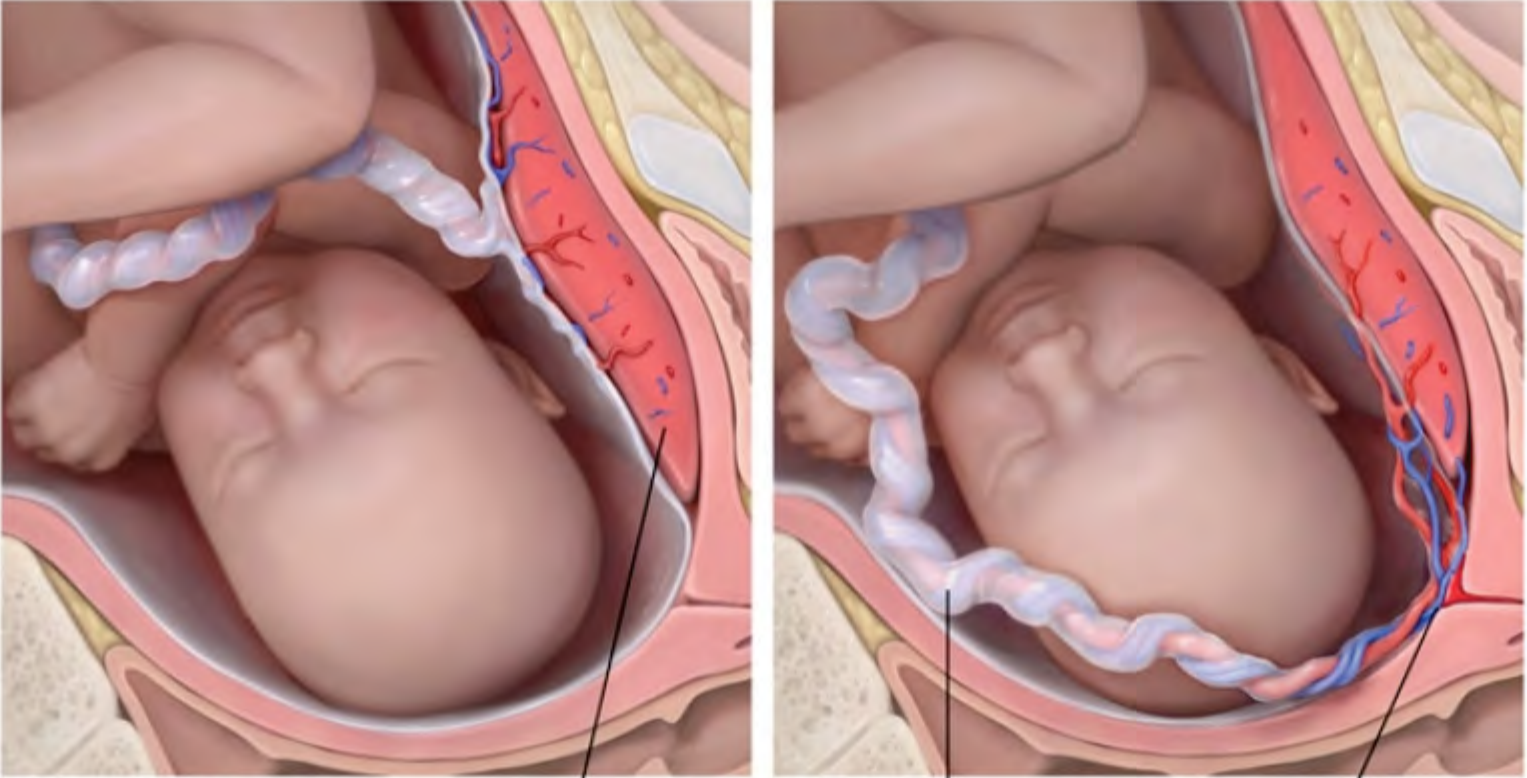
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This patient in the third trimester with **painful vaginal bleeding** and a **tender, firm uterus** has a presentation

Exhibit Display

Normal anatomy

Vasa previa



Placenta

Fetal vessels within Wharton jelly

Unprotected vessels at internal os

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Educational objective:

Abruptio placentae, detachment of the placenta from the uterus prior to fetal delivery, presents with painful vaginal

cocaine (vasoconstrictors causing placental ischemia), and prior abruptio placentae.

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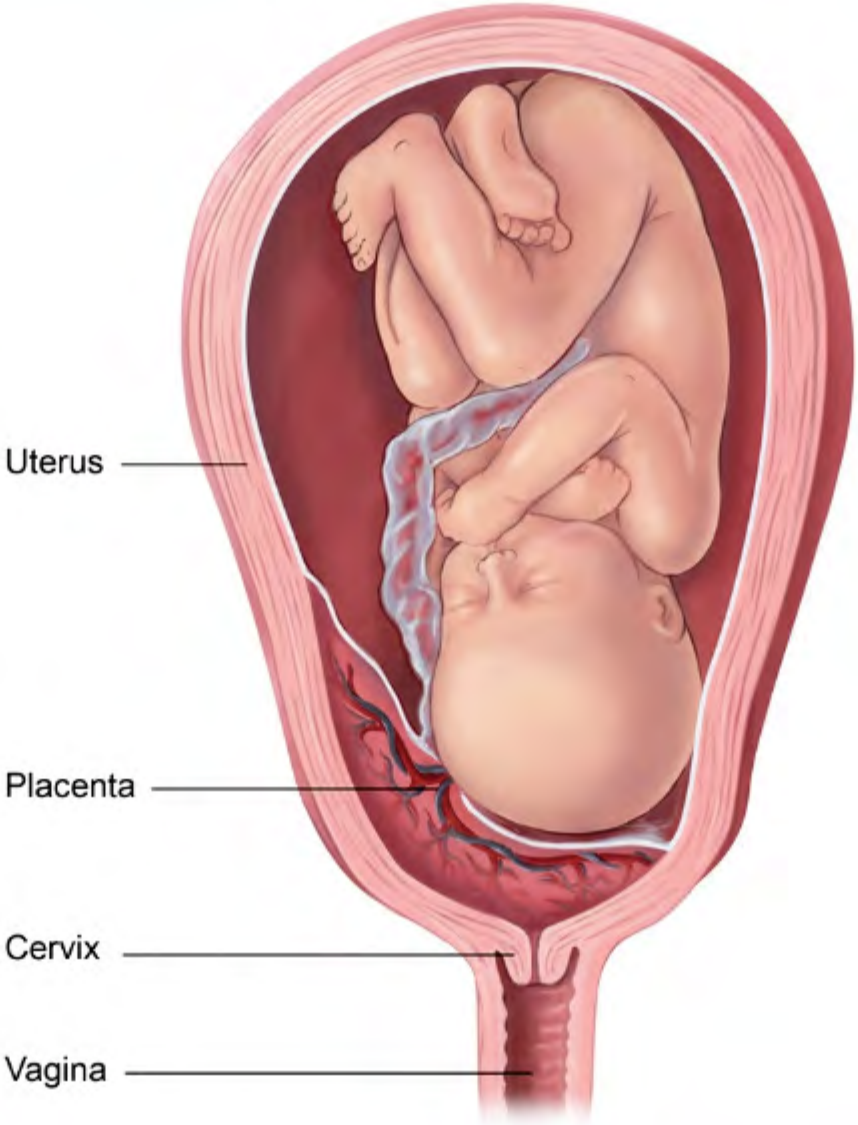
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hypertension, and tobacco or cocaine use.

Exhibit Display

Placenta previa



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A 2-day-old girl is evaluated in the newborn nursery due to tachypnea, sneezing, and diarrhea. The patient was born via spontaneous vaginal delivery and had been feeding well until several hours ago, when she became tachypneic. She has also been persistently crying and difficult to console. The patient's mother did not receive prenatal care during this pregnancy. Respirations are 65/min. All other vital signs are normal for age. On physical examination, the patient is irritable and has mildly increased tone in all extremities with occasional tremors of the arms and legs. Chest radiograph reveals normal lung fields. Serum glucose and complete blood count are within normal limits, and urine toxicology is pending. Nonpharmacologic interventions do not significantly improve the patient's symptoms. Which of the following pharmacotherapies would most likely improve this patient's symptoms?

- ☐ A. Flumazenil

☐ B. Morphine

☐ C. Naloxone

☐ D. Sodium bicarbonate

☐ E. Vitamin K

Submit

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- A. Flumazenil (10%)

✓

B. Morphine (36%)

✗

C. Naloxone (32%)

D. Sodium bicarbonate (12%)

E. Vitamin K (8%)

Incorrect

Correct answer
B

36%
Answered correctly

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Explanation

Neonatal abstinence syndrome	
Pathophysiology	<ul style="list-style-type: none">Withdrawal from transplacental opiates due to maternal drug use

Neonatal abstinence syndrome	
Pathophysiology	<ul style="list-style-type: none">Withdrawal from transplacental opiates due to maternal drug use
Clinical manifestations	<ul style="list-style-type: none">Neurologic: irritability, hypertonia, jittery movements, seizures (rare)Gastrointestinal: diarrhea, vomiting, feeding intoleranceAutonomic: sweating, sneezing, pupillary dilation
Treatment	<ul style="list-style-type: none">Opioid therapy (eg, morphine, methadone)

This newborn has signs of **neonatal abstinence syndrome (NAS)**, or withdrawal from transplacental exposure to maternal substances (primarily opioids). NAS due to **opioid withdrawal** occurs in the setting of maternal prescription use/misuse, illicit drug use, or treatment for a substance use disorder (eg, methadone). Newborns exposed to opioids with shorter half-lives (eg, heroin) are symptomatic sooner (eg, age 1-2 days) than those exposed to drugs with longer half-lives (eg, methadone).

Characteristic symptoms reflect a dysregulated CNS and autonomic dysfunction. **Excessive irritability**, often caused by hyperresponsiveness to external stimuli (eg, bright lights, loud noise), and short, interrupted sleep cycles are typical. Symptoms of autonomic dysfunction include sweating, **sneezing**, and yawning. In addition, **diarrhea**, **vomiting**, and poor feeding are common. Examination often reveals **tachypnea**, **tremors**, and **hypertonia**, as seen in this patient.

The initial treatment for NAS is **nonpharmacologic** modifications, such as a low-stimuli environment (eg, quiet room) and swaddling to reduce tremors and irritability. Symptoms refractory to nonpharmacologic treatment, as in this case, warrant **opioid replacement** (eg, morphine, methadone). Once symptoms are controlled, the medication is weaned off over several weeks.

(Choice A) Neonatal withdrawal to maternal benzodiazepine use (less common than opioid use) can present similarly to opioid withdrawal and may warrant pharmacotherapy (eg, phenobarbital). However, flumazenil, a GABA receptor antagonist used for benzodiazepine reversal, can worsen benzodiazepine withdrawal.

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(Choice C) Naloxone is an opioid receptor antagonist used for acute opioid overdose. Administering naloxone for opioid withdrawal can result in more severe withdrawal symptoms and is contraindicated.

(Choice D) Sodium bicarbonate can be used to treat toxic ingestions of tricyclic antidepressants and salicylates. However, toxic maternal ingestion of these substances would present with neonatal symptoms at birth, not two days later.

(Choice E) Vitamin K prevents and treats vitamin K deficiency bleeding, which can occur in newborns with

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(Choice E) Vitamin K prevents and treats vitamin K deficiency bleeding, which can occur in newborns with mothers who take medications that block vitamin K (eg, anticonvulsants). Unlike this case, patients develop bleeding (eg, mucosal surfaces, gastrointestinal tract, CNS).

Educational objective:

Neonatal abstinence syndrome due to maternal opioid use causes neurologic, gastrointestinal, and autonomic symptoms; irritability, tremors, diarrhea, and sneezing are classic findings. Patients with symptoms refractory to nonpharmacologic therapy warrant opioid replacement (eg, morphine).

A 32-year-old woman, gravida 2 para 1, at 30 weeks gestation comes to the office for a routine prenatal visit. The patient's pregnancy has been uncomplicated, and she has had no vaginal bleeding or contractions. She has continued to jog daily throughout the pregnancy but is becoming more fatigued by the end of her workouts. The patient has no chronic medical conditions, and her only medication is a daily prenatal vitamin. She does not use tobacco, alcohol, or illicit drugs. Blood pressure is 110/70 mm Hg, pulse is 76/min, and respirations are 20/min. Fetal heart tones are normal. Physical examination is unremarkable. Compared to a nonpregnant state, this patient most likely has which of the following hematologic changes?

	Plasma volume	Red blood cell mass	Hemoglobin concentration
<input type="radio"/> A.	↑	↑	↓
<input type="radio"/> B.	↑	↓	↓
<input type="radio"/> C.	↓	↑	↑
<input type="radio"/> D.	No change	↑	↑
<input type="radio"/> E.	No change	↓	↓

Submit

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	Plasma volume	Red blood cell mass	Hemoglobin concentration	
✓ <input type="radio"/> A.	↑	↑	↓	(63%)
✗ <input checked="" type="radio"/> B.	↑	↓	↓	(27%)
<input type="radio"/> C.	↓	↑	↑	(2%)
<input type="radio"/> D.	No change	↑	↑	(4%)
<input type="radio"/> E.	No change	↓	↓	(1%)

Incorrect

Correct answer
A

63%
Answered correctly

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Explanation

Physiologic changes of pregnancy

Physiologic changes of pregnancy	
Cardiovascular	<ul style="list-style-type: none">• ↑ Blood volume (plasma > RBC mass)• ↓ Systemic vascular resistance• ↑ Heart rate & cardiac output
Pulmonary	<ul style="list-style-type: none">• ↑ Central respiratory drive (hyperventilation)• ↓ PaCO₂ (respiratory alkalosis), ↑ PaO₂
Renal	<ul style="list-style-type: none">• ↑ Renal blood flow & urine output• ↑ GFR, ↓ BUN & serum creatinine• ↑ HCO₃⁻ excretion (metabolic compensation)• ↓ Serum Na⁺ concentration (↑ ADH secretion)
Hematologic	<ul style="list-style-type: none">• ↑ Prothrombotic coagulation factors• ↓ Hemoglobin concentration (dilutional anemia)
ADH = antidiuretic hormone; BUN = blood urea nitrogen; GFR = glomerular filtration rate; RBC = red blood cell.	

During **normal pregnancy**, the maternal hematologic system undergoes several adaptations to accomodate the developing fetus and placenta. These adaptations include:

- **Increased plasma volume**, which begins at approximately 6 weeks gestation and increases rapidly until term, resulting in a total plasma volume increase of 30%-50% when compared to a nonpregnant state. Increased plasma volume helps reduce positional hypotension caused by uterine compression of the vena cava (ie, decreased venous return when patient is supine or standing) and ensures adequate circulating volume for fetal nutrient delivery and waste removal.
- **Increased maternal red blood cell mass**. Pregnant women generate higher levels of erythropoietin, which drives the production of new red blood cells and increases red blood cell mass by 20%-30%. This increase

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- **Increased maternal red blood cell mass**. Pregnant women generate higher levels of erythropoietin, which drives the production of new red blood cells and increases red blood cell mass by 20%-30%. This increase helps ensure adequate oxygen delivery to the fetus and placenta; it also **protects against excessive blood loss** associated with delivery (eg, postpartum hemorrhage).

Because the expansion of maternal blood plasma volume is greater than the increase in maternal red blood cell mass, pregnant women typically have a **mild reduction in hemoglobin concentration** due to dilution.

Educational objective:

Pregnant patients have marked increases in blood plasma volume (by 30%-50%) and red blood cell mass (by 20%-30%), which helps improve fetal nutrient delivery while also protecting against excessive blood loss during delivery. Because the expansion of blood plasma volume is greater than the increase in red blood cell mass, pregnant women typically have mildly decreased hemoglobin concentrations (ie, dilutional anemia).

References

- [Hematologic complications of pregnancy.](#)

A 27-year-old nulligravid woman comes to the office for a routine visit. Medical history is significant for epilepsy that is well controlled with valproate and chronic hypertension for which she takes nifedipine. She hopes to conceive in the next few months and plans to start prenatal vitamins soon. The patient takes no other medications and does not use tobacco, alcohol, or illicit drugs. Blood pressure is 120/80 mm Hg. Physical examination is normal. If the patient conceives now, her fetus is at increased risk for which of the following anomalies?

- ☐ A. Epiphyseal stippling
- ☐ B. Hypothyroidism
- ☐ C. Micrognathia
- ☐ D. Myelomeningocele
- ☐ E. Nasal hypoplasia
- ☐ F. Renal dysgenesis
- ☐ G. Tricuspid valve malformation

Submit

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- A. Epiphyseal stippling (1%)
- B. Hypothyroidism (1%)
- ✗

☒ C. Micrognathia (3%)
- ✓

☐ D. Myelomeningocele (79%)
- E. Nasal hypoplasia (1%)
- F. Renal dysgenesis (6%)
- G. Tricuspid valve malformation (5%)

Incorrect

Correct answer
D

79%
Answered correctly

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Explanation

Teratogenic medications	
Drug	Adverse effects
	Neural tube defects, microcephaly, orofacial

Teratogenic medications	
Drug	Adverse effects
Phenytoin	Neural tube defects, microcephaly, orofacial clefts, dysmorphic facial features, distal digit/nail hypoplasia
Lithium	Ebstein anomaly, nephrogenic diabetes insipidus, hypothyroidism
Valproate	Neural tube defects
Isotretinoin	Microcephaly, thymic hypoplasia, small ears, hydrocephalus
Methotrexate	Limb & craniofacial abnormalities, neural tube defects, abortion
ACE inhibitors	Renal dysgenesis, oligohydramnios
Warfarin	Nasal hypoplasia, stippled epiphysis

Preconception counseling is recommended in reproductive-age women with chronic medical conditions and desire for pregnancy to optimize the patient's health and identify risk factors for potential obstetric complications.

This patient with epilepsy controlled with **valproate** is at increased risk for fetal **neural tube defects**. Valproate, an antiepileptic agent also used to prevent migraine headache and treat bipolar disorder, is **contraindicated in pregnancy** because it interferes with folate metabolism and causes **maternal folate deficiency**. Folic acid is required to regulate fetal neural tube closure in early pregnancy; therefore, patients taking valproate are at increased risk for incomplete neural tube closure (eg, meningocele, [myelomeningocele](#)) compared to the general

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Because most women discover that they are pregnant after the most vulnerable period of neural tube formation (eg, 4 weeks gestation), patients on valproate should try to optimize seizure control prior to conception with an **alternate anticonvulsant** (eg, levetiracetam). Low-dose **folic acid supplementation** is recommended in all women before conception; high-dose folic acid supplementation is recommended in the presence of risk factors such as current valproate use or a prior child with a neural tube defect.

(Choices A and E) Epiphyseal stippling (pinpoint calcification) and nasal hypoplasia (depression of the nasal bridge) are characteristics of warfarin embryopathy caused by first-trimester exposure to warfarin.

(Choices B and G) Maternal hypothyroidism, neonatal hypothyroidism, and fetal tricuspid valve malformation (ie, [Ebstein anomaly](#)) are associated with lithium use during pregnancy. Neonatal hypothyroidism can also result from exposure to maternal antithyroid medications (eg, propylthiouracil, methimazole).

(Choice C) Micrognathia, or hypoplasia of the mandible, is a craniofacial malformation that can occur with fetal exposure to isotretinoin. It is also commonly associated with genetic syndromes (eg, [Trisomy 18](#)).

(Choice F) Renal dysgenesis and fetal anuria/oligohydramnios can result from the use of ACE inhibitors and angiotensin receptor blockers during pregnancy. These medications are contraindicated in pregnancy, but other antihypertensive medications such as nifedipine and labetalol are safe because they have no teratogenic effects.

antiepileptic agent also used to prevent migraine headache and treat bipolar disorder, is **contraindicated in pregnancy** because it interferes with folate metabolism and causes **maternal folate deficiency**. Folic acid is required to regulate fetal neural tube closure in early pregnancy; therefore, patients taking valproate are at increased risk for incomplete neural tube closure (eg, meningocele, [myelomeningocele](#)) compared to the general population.

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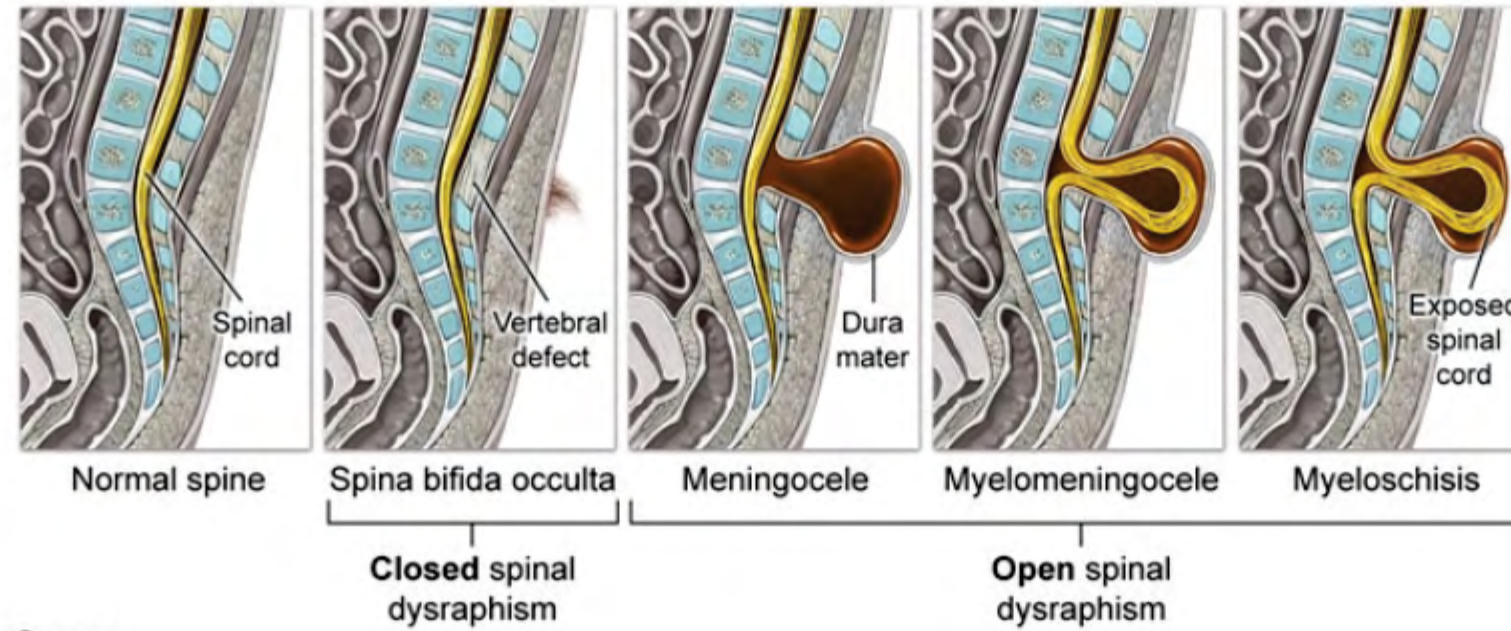
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Educational objective:

Valproate is a teratogenic medication that decreases maternal folate levels and increases the risk of fetal neural tube defects (eg, meningocele, myelomeningocele).

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Spina bifida

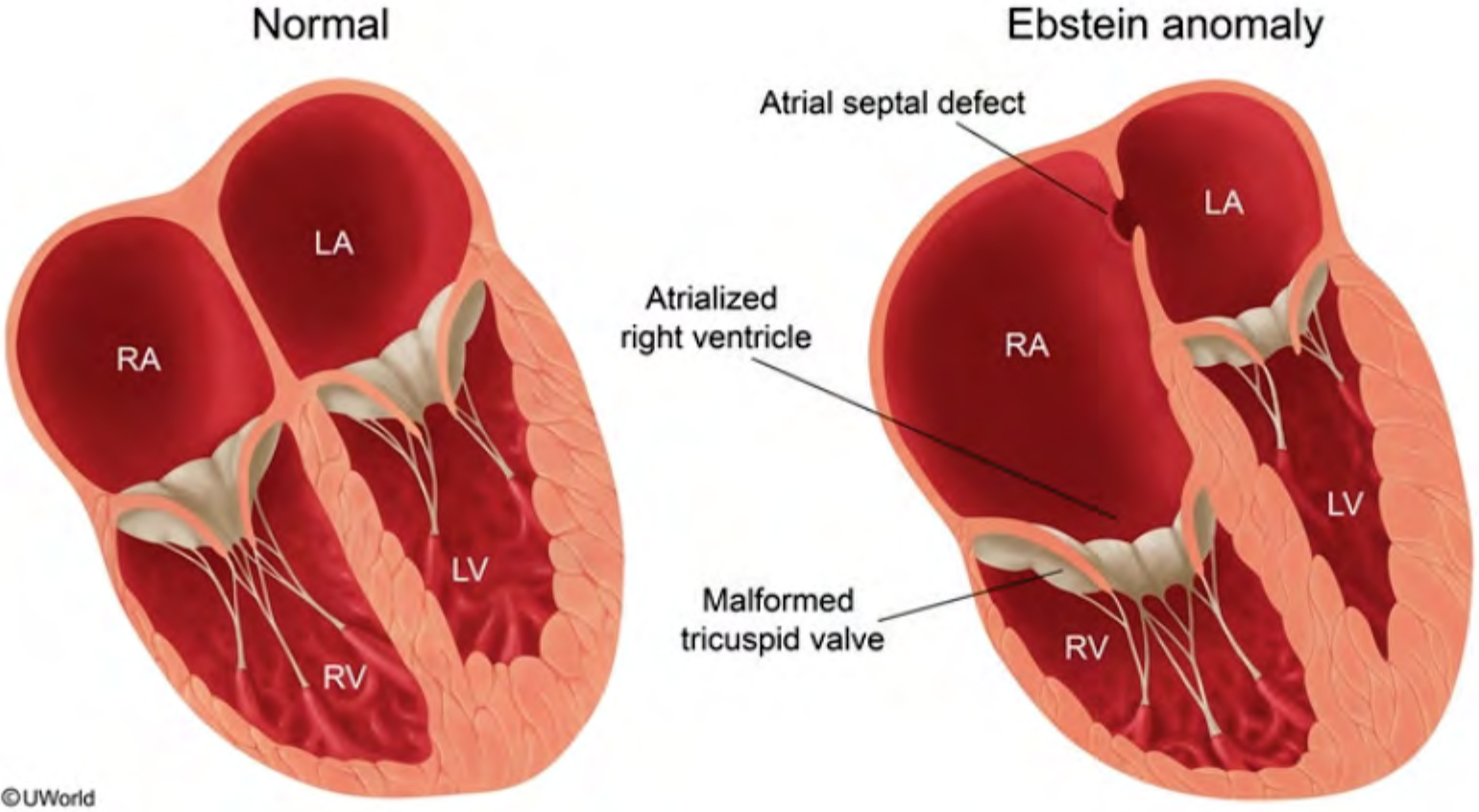


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antiepileptic agent also used to prevent migraine headache and treat bipolar disorder, is **contraindicated** in

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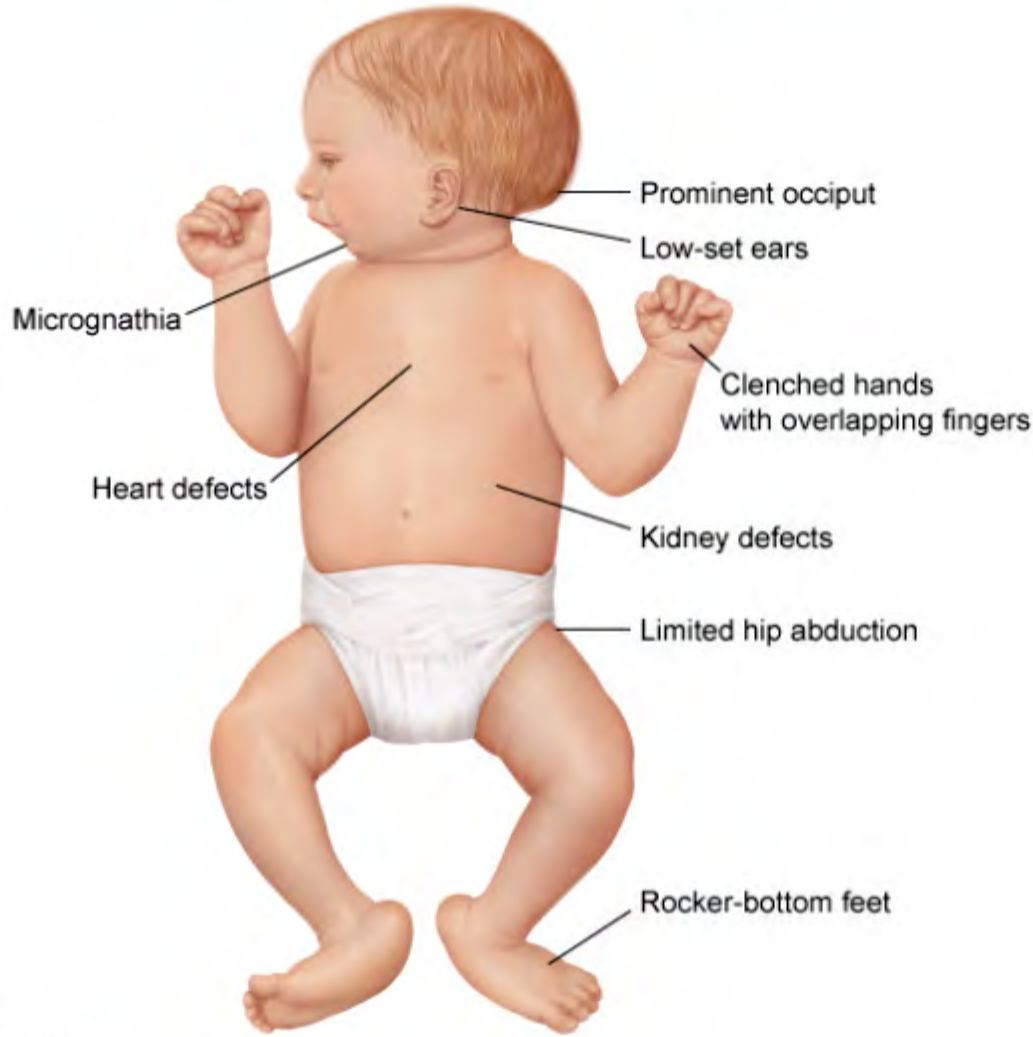


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antiepileptic agent also used to prevent migraine headache and treat bipolar disorder, is **contraindicated** in

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Trisomy 18 (Edwards syndrome)



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Valproate is a teratogenic medication that decreases maternal folate levels and increases the risk of fetal neural tube defects (eg, meningocele, myelomeningocele).

A 29-year-old woman, gravida 2 para 1, at 32 weeks gestation comes to the emergency department due to heavy vaginal bleeding. The bleeding started an hour ago. It was light initially and associated with only mild abdominal pain, but both the bleeding and the pain have increased, and now the pain is constant and severe. The patient has no chronic medical conditions. Her prior pregnancy was a term vaginal delivery complicated by preeclampsia with severe features. Blood pressure is 156/98 mm Hg, and pulse is 112/min. The uterus is firm and tender. Pelvic examination reveals heavy bleeding from the cervical os. Which of the following is the most likely cause of this patient's current presentation?

- ☐ A. Abnormal invasion of trophoblast into uterine myometrium
- ☐ B. Bleeding placental tissue extending over the cervix
- ☐ C. Physiologic cervical dilation due to spontaneous labor
- ☐ D. Premature separation of the placenta and myometrium
- ☐ E. Rupture of the uterine myometrium and serosa

Submit

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- ✖

A. Abnormal invasion of trophoblast into uterine myometrium (4%)
- B. Bleeding placental tissue extending over the cervix (9%)
- C. Physiologic cervical dilation due to spontaneous labor (0%)
- ✔

D. Premature separation of the placenta and myometrium (81%)
- E. Rupture of the uterine myometrium and serosa (3%)

Incorrect

Correct answer
D

81%
Answered correctly

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Explanation

Abruptio placentae	
Definition	<ul style="list-style-type: none">Premature placental separation from uterus
Risk factors	<ul style="list-style-type: none">Hypertension, preeclampsia

Abruptio placentae	
Definition	<ul style="list-style-type: none">• Premature placental separation from uterus
Risk factors	<ul style="list-style-type: none">• Hypertension, preeclampsia• Abdominal trauma• Cocaine or tobacco use• Prior abruptio placentae
Clinical features	<ul style="list-style-type: none">• Sudden-onset vaginal bleeding• Abdominal pain• High-frequency contractions• Tender, firm uterus

This patient in the third trimester with **painful vaginal bleeding** likely has **abruptio placentae**, the premature separation of the placenta from the myometrium prior to fetal delivery. **Placental abruption** occurs when **maternal vessels rupture** at the uteroplacental interface (ie, decidua basalis); the resultant bleeding causes **placental separation** from the uterine myometrium. Blood can also accumulate between the placenta and uterine wall and cause an acute increase in intrauterine pressure, resulting in severe abdominal pain, uterine irritability (ie, high-frequency contractions), and a **tender, firm uterus**.

In this patient, abruptio placentae is likely a complication of **preeclampsia**, a hypertensive disorder of pregnancy that causes widespread **endothelial cell damage**. Endothelial cell dysfunction results in dysregulated vascular tone (eg, hypertension) and **increased vessel fragility**. Preeclampsia also impairs early spiral artery development needed to supply blood to the fetus and placenta. As a result, patients with preeclampsia have abnormally high-resistance spiral arteries that produce low placental perfusion, ischemia, and possible placental infarction, all of which increase the risk of abruptio placentae.

(Choice A) The abnormal invasion of trophoblast into uterine myometrium (eg, placenta accreta) can cause heavy

In this patient, abruptio placentae is likely a complication of **preeclampsia**, a hypertensive disorder of pregnancy that causes widespread **endothelial cell damage**. Endothelial cell dysfunction results in dysregulated vascular tone (eg, hypertension) and **increased vessel fragility**. Preeclampsia also impairs early spiral artery development needed to supply blood to the fetus and placenta. As a result, patients with preeclampsia have abnormally high-resistance spiral arteries that produce low placental perfusion, ischemia, and possible placental infarction, all of which increase the risk of abruptio placentae.

(Choice A) The abnormal invasion of trophoblast into uterine myometrium (eg, [placenta accreta](#)) can cause heavy vaginal bleeding. However, this disorder is typically diagnosed after fetal delivery when the placenta does not detach from the uterine wall, resulting in postpartum (not antepartum) hemorrhage.

(Choice B) [Placenta previa](#), the extension of placental tissue over the cervix, can cause antepartum bleeding. However, placental bleeding from this location can readily exit the uterus via the cervix, so patients typically do not have severe abdominal pain or a firm, tender uterus.

(Choice C) Physiologic cervical dilation (ie, spontaneous labor) can progress from light vaginal bleeding with mild abdominal pain to heavier bleeding during contractions. Labor pain, however, is typically intermittent, and the uterus relaxes between contractions.

(Choice E) [Rupture](#) of the uterine myometrium and serosa can cause abdominal pain and heavy vaginal bleeding. However, in the classic presentation, fetal parts are palpable through the maternal abdominal wall and patients do not have a firm uterus. In addition, this patient has no risk factors (eg, prior cesarean delivery), making this diagnosis less likely.

Educational objective:

Abruptio placentae is caused by rupture of maternal vessels at the uteroplacental interface that leads to premature separation of the placenta from the myometrium. Patients typically have painful vaginal bleeding and a tender, firm uterus. Preeclampsia increases the risk of abruptio placentae.

This patient in the third trimester with **painful vaginal bleeding** likely has **abruptio placentae**, the premature

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Abruptio placentae

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patients do not have a firm uterus. In addition, this patient has no risk factors (eg, prior cesarean delivery), making

This patient in the third trimester with **painful vaginal bleeding** likely has **abruptio placentae**, the premature

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Placenta previa

Uterus

Placenta

Cervix

Vagina

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separation of the placenta from the uterine wall. This condition is often associated with **painful vaginal bleeding** in the third trimester. In the classic presentation, fetal parts are palpable through the maternal abdominal wall and patients do not have a firm uterus. In addition, this patient has no risk factors (eg, prior cesarean delivery), making

separation from the uterine myometrium. Blood can also accumulate between the placenta and uterine wall and

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Placenta accreta

The diagram illustrates the spectrum of placenta accreta. On the left, a sagittal view of a pregnant uterus shows the placenta's location. An arrow points to a detailed cross-section of the uterine wall. The layers are labeled: Endometrium (top), Myometrium (middle muscle layer), and Serosa (outer layer). The diagram is divided into four vertical sections representing different types of placental attachment:

- Normal decidua:** Shows a clear boundary between the placenta and the underlying myometrium.
- Accreta (63%):** Shows the placenta invading the myometrium, with no visible decidua.
- Increta (15%):** Shows the placenta invading deeper into the myometrium.
- Percreta (22%):** Shows the placenta invading through the myometrium and into the serosa.

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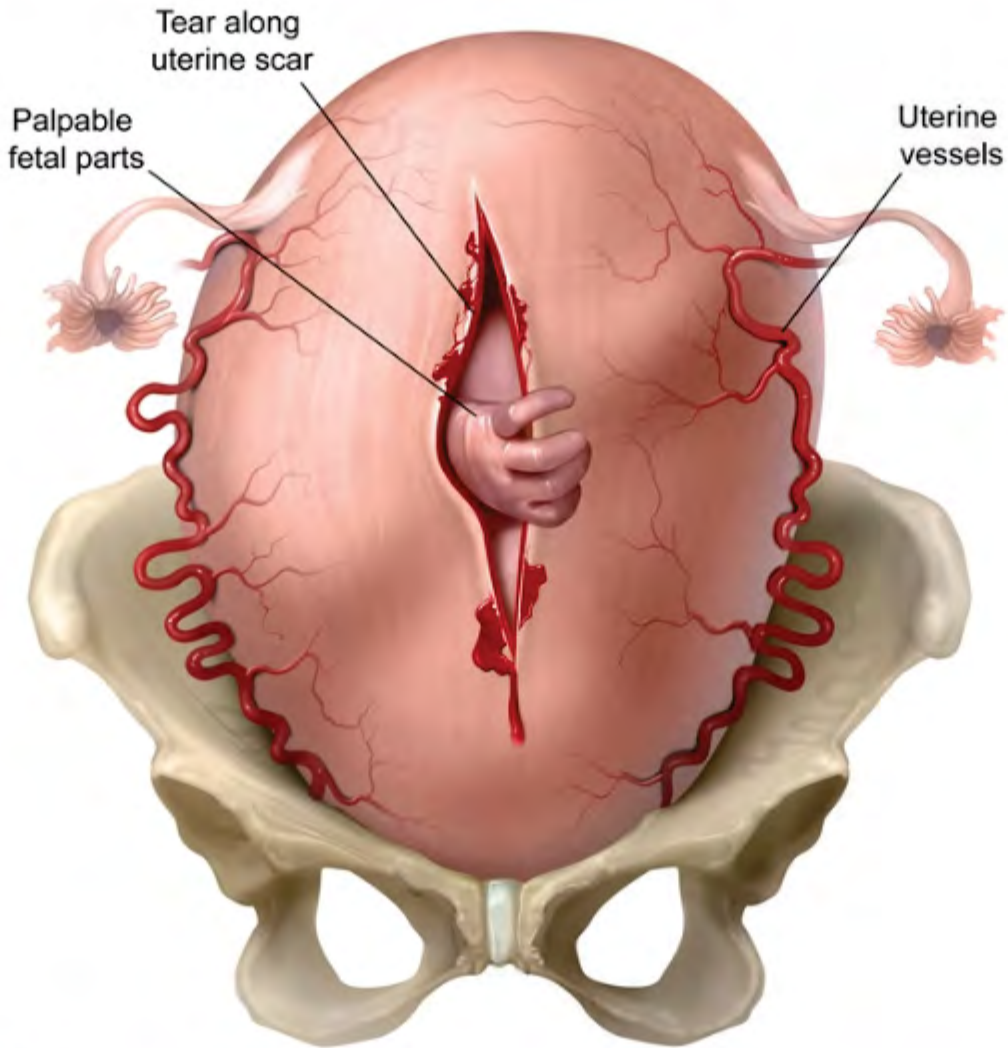
Educational objective:

Abruption placentae is caused by rupture of maternal vessels at the uteroplacental interface that leads to premature

separation from the uterine myometrium. Blood can also accumulate between the placenta and uterine wall and

Exhibit Display

Uterine rupture



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Educational objective:

Abruption placentae is caused by rupture of maternal vessels at the uteroplacental interface that leads to premature

A 19-year-old woman, gravida 1 para 0, at 29 weeks gestation comes to the emergency department due to severe right upper quadrant pain. The patient suddenly developed the pain this evening after dinner. The pain was associated with acute nausea and vomiting. She has no headaches or visual changes. The patient has no chronic medical conditions and takes no daily medications. Temperature is 36.7 C (98.1 F), blood pressure is 160/100 mm Hg, and pulse is 84/min. The abdomen is tender over the right upper quadrant, but no rebound or guarding is present. Hemoglobin is 8 g/dL. Urinalysis shows 3+ protein. Which of the following associated laboratory changes are most likely to be seen in this patient?

	Alanine aminotransferase	Indirect bilirubin	Platelets
<input type="radio"/> A.	↑	↑	↓
<input type="radio"/> B.	↑	Normal	Normal
<input type="radio"/> C.	↑	↓	Normal
<input type="radio"/> D.	Normal	↑	↓
<input type="radio"/> E.	Normal	Normal	↓

Submit

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	Alanine aminotransferase	Indirect bilirubin	Platelets	
<div><div>✓</div><div><input type="radio"/></div></div> A.	↑	↑	↓	(71%)
<div><div>✗</div><div><input checked="" type="radio"/></div></div> B.	↑	Normal	Normal	(12%)
<div><div></div><div><input type="radio"/></div></div> C.	↑	↓	Normal	(4%)
<div><div></div><div><input type="radio"/></div></div> D.	Normal	↑	↓	(7%)
<div><div></div><div><input type="radio"/></div></div> E.	Normal	Normal	↓	(4%)

Incorrect

Correct answer
A

71%
Answered correctly

05 secs
Time Spent

2023
Version

Explanation

HELLP syndrome

HELLP syndrome	
Pathogenesis	<ul style="list-style-type: none">Abnormal placental developmentMaternal endothelial dysfunction
Clinical features	<ul style="list-style-type: none">HypertensionRight upper quadrant painNausea/vomitingPossible headache or vision changes
Laboratory findings	<ul style="list-style-type: none">Microangiopathic hemolytic anemiaThrombocytopeniaElevated liver enzymes & bilirubinProteinuria
HELLP = Hemolysis, Elevated Liver enzymes, Low Platelet count.	

This patient at >20 weeks gestation with hypertension, proteinuria, and right upper quadrant pain likely has **HELLP** (**H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelet count) **syndrome**. HELLP syndrome is most often diagnosed in patients with **preeclampsia**, which likely occurs due to abnormal placental development ([insufficient cytotrophoblast invasion](#) in early pregnancy) and subsequent maternal endothelial dysfunction.

Unlike preeclampsia, HELLP syndrome is associated with excessive activation of the coagulation cascade, likely because of severe endothelial injury. This causes widespread thrombi formation in the microvasculature (ie, **microangiopathic thrombotic microangiopathy**). As platelets are consumed and numerous thrombi form, patients develop severe **thrombocytopenia** and mechanical shearing of circulating red blood cells, resulting in **hemolytic anemia**.

A hallmark of both preeclampsia and HELLP syndrome is **hepatocellular injury** (due to impaired hepatic perfusion), which leads to **increased alanine aminotransferase** release, **increased indirect bilirubin**

HELLP = Hemolysis, Elevated Liver enzymes, Low Platelet count.

This patient at >20 weeks gestation with hypertension, proteinuria, and right upper quadrant pain likely has **HELLP** (**H**emolysis, **E**levated **L**iver enzymes, **L**ow **P**latelet count) **syndrome**. HELLP syndrome is most often diagnosed in patients with **preeclampsia**, which likely occurs due to abnormal placental development ([insufficient cytotrophoblast invasion](#) in early pregnancy) and subsequent maternal endothelial dysfunction.

Unlike preeclampsia, HELLP syndrome is associated with excessive activation of the coagulation cascade, likely because of severe endothelial injury. This causes widespread thrombi formation in the microvasculature (ie, **microangiopathic thrombotic microangiopathy**). As platelets are consumed and numerous thrombi form, patients develop severe **thrombocytopenia** and mechanical shearing of circulating red blood cells, resulting in **hemolytic anemia**.

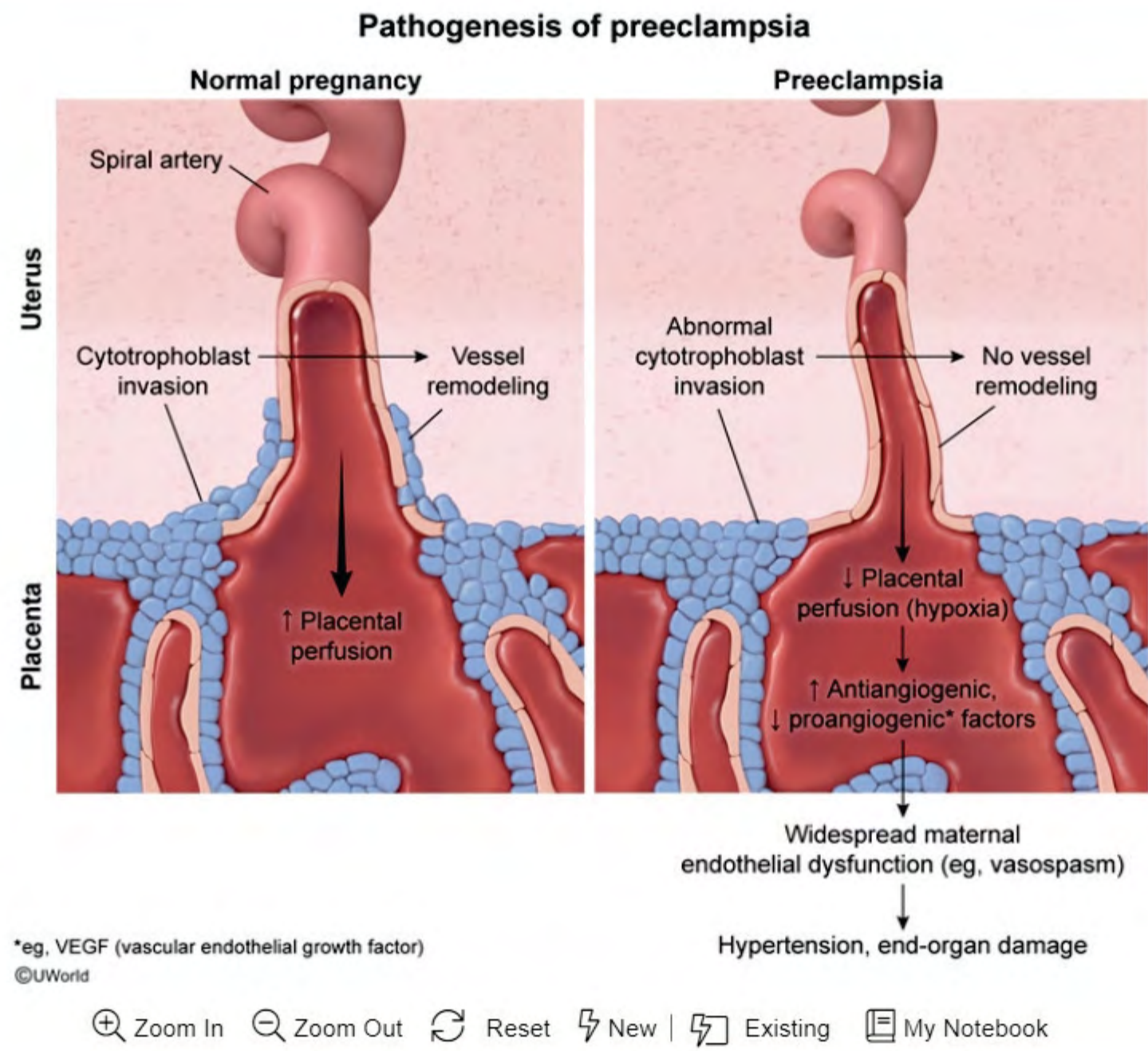
A hallmark of both preeclampsia and HELLP syndrome is **hepatocellular injury** (due to impaired hepatic perfusion), which leads to **increased alanine aminotransferase** release, **increased indirect bilirubin** accumulation (exacerbated by the ongoing hemolysis), and clinical features of **right upper quadrant pain** with nausea/vomiting. More serious complications include liver necrosis, hepatic hemorrhage, subcapsular hematoma, and liver rupture.

Educational objective:

HELLP (Hemolysis, Elevated Liver enzymes, Low Platelet count) syndrome is a thrombotic microangiopathic disorder that causes microangiopathic hemolytic anemia (eg, increased indirect bilirubin levels), thrombocytopenia, and hepatocellular injury (eg, increased alanine aminotransferase). Patients may have hypertension and right upper quadrant pain with nausea/vomiting.

HELLP = Hemolysis. Elevated Liver enzymes. Low Platelet count.

Exhibit Display



A 31-year-old woman, gravida 2 para 1, at 30 weeks gestation comes to the emergency department due to increased swelling in her face and lower extremities. She has noticed a sudden onset of swelling and a 2.72-kg (6-lb) weight gain in the last day. The patient has not urinated in the last 12 hours despite an increase in fluid intake. She has a twin pregnancy and no chronic medical conditions. Blood pressure is 154/98 mm Hg, and pulse is 98/min. Bladder catheterization yields a total of 10 mL of dark urine. Urinalysis shows no red blood cells. Which of the following pathologic factors is the most likely cause of this patient's decreased urine output?

- ☐ A. Inflammation of the glomeruli
- ☐ B. Immune complex deposition within glomeruli
- ☐ C. Inflammation of the renal interstitium
- ☐ D. Obstructing stone within the renal calyx
- ☐ E. Vasospasm of the intrarenal arteries

Submit

A 31-year-old woman, gravida 2 para 1, at 30 weeks gestation comes to the emergency department due to increased swelling in her face and lower extremities. She has noticed a sudden onset of swelling and a 2.72-kg (6-lb) weight gain in the last day. The patient has not urinated in the last 12 hours despite an increase in fluid intake. She has a twin pregnancy and no chronic medical conditions. Blood pressure is 154/98 mm Hg, and pulse is 98/min. Bladder catheterization yields a total of 10 mL of dark urine. Urinalysis shows no red blood cells. Which of the following pathologic factors is the most likely cause of this patient's decreased urine output?

- ✗

☒ A. Inflammation of the glomeruli (12%)
- ☐ B. Immune complex deposition within glomeruli (13%)
- ☐ C. Inflammation of the renal interstitium (12%)
- ☐ D. Obstructing stone within the renal calyx (13%)
- ✓

☐ E. Vasospasm of the intrarenal arteries (47%)

Incorrect

Correct answer
E

 47%

Answered correctly

 07 secs

Time Spent

 2023

Version

Explanation

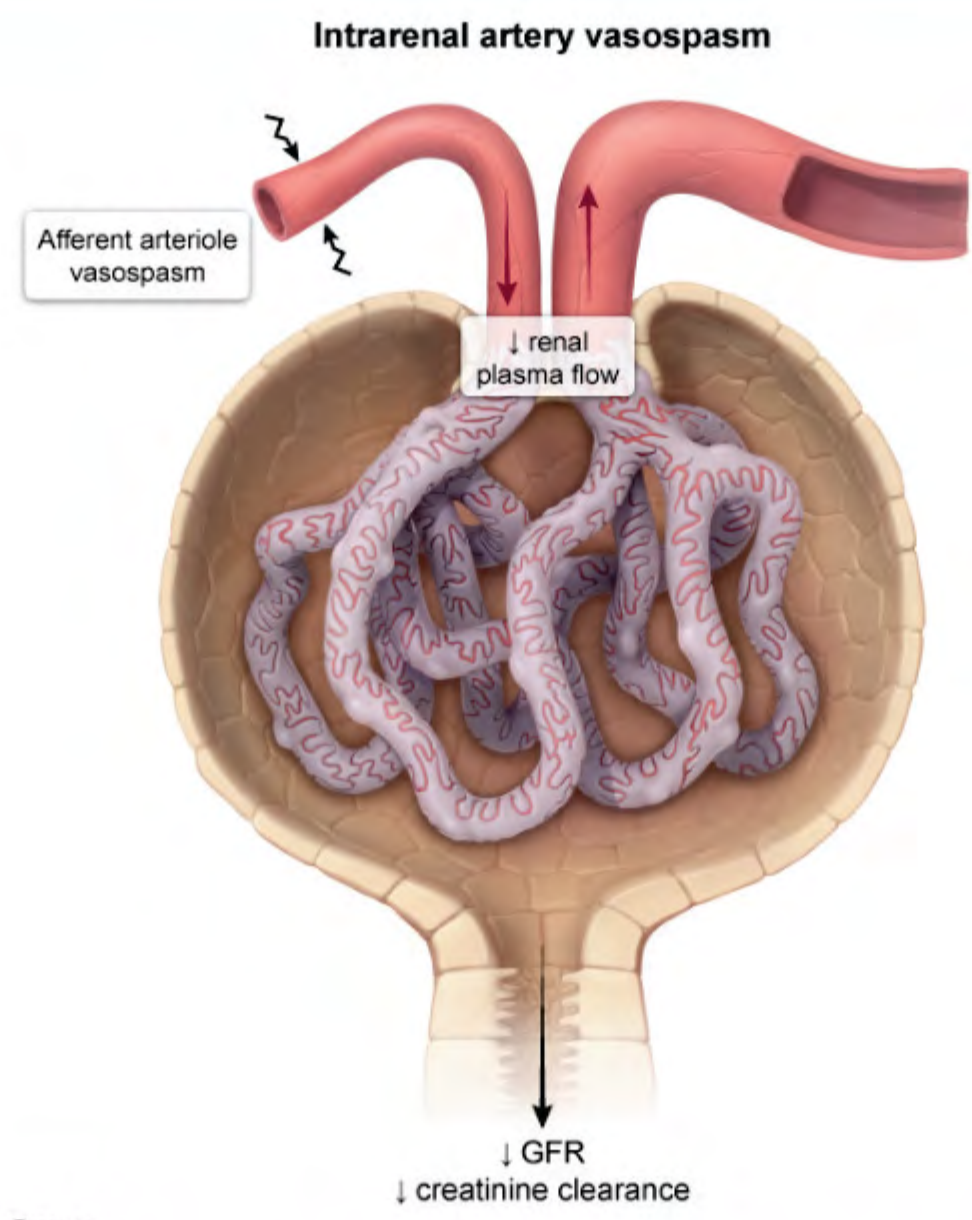


A 31-year-old woman, gravida 2, para 1, at 20 weeks gestation comes to the emergency department due to

increased blood pressure.
Her blood pressure is 160/100 mm Hg.
She has no other symptoms.

- A. She has gestational hypertension.
- B. She has chronic hypertension.
- C. She has preeclampsia.
- D. She has eclampsia.
- E. She has a urinary tract infection.

Exhibit Display



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This patient at 30 weeks gestation has **oliguria**, or decreased urine output (<500 mL/24 hr), likely due to preeclampsia. **Preeclampsia** is defined as **new-onset hypertension** (systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg) at ≥ 20 weeks gestation plus proteinuria and/or signs of **end-organ dysfunction** (eg, renal failure).

Preeclampsia is thought to be caused by abnormal placentation and poorly developed uterine spiral arteries, which lead to **placental ischemia**. Twin gestations are at increased risk, likely due to increased placental mass.

In preeclampsia, the ischemic placenta releases **antiangiogenic factors** that cause widespread **maternal endothelial cell damage**. The resulting **capillary leakage** is responsible for third spacing of fluid (eg, facial edema, weight gain) and leakage of protein into the urine (ie, proteinuria). In addition, disrupted endothelial control of vascular tone causes hypertension and **widespread vasospasm**, resulting in end-organ hypoperfusion. In the kidneys, vasospasm causes decreased renal blood flow and glomerular filtration rate, leading to **minimal, concentrated urine** (ie, high specific gravity) and increased serum creatinine levels.

Gradual recovery of renal function typically occurs following delivery of the placenta, which is curative for preeclampsia.

(Choices A and B) Inflammation of the glomeruli (ie, glomerulonephritis) can occur as a result of glomerular immune complex deposition, such as with anti-glomerular basement membrane (Goodpasture) disease (ie, basement membrane IgG deposits) and IgA nephropathy (ie, mesangial IgA deposits). Although these conditions can cause nephritic syndrome (eg, hypertension, mild to moderate proteinuria and edema, oliguria), red blood cells and casts would be expected on urinalysis.

(Choice C) Inflammation of the renal interstitium (eg, acute interstitial nephritis) can cause oliguria but is typically drug related (eg, nonsteroidal anti-inflammatory drugs, beta-lactam antibiotics) and associated with rash and fever, which are not seen in this patient.

(Choice D) A stone within the renal calyx (eg, staghorn calculus) may decrease the urine output of the affected kidney due to obstruction; however, overall urine output is typically preserved due to normal functioning of the

In preeclampsia, the ischemic placenta releases **antiangiogenic factors** that cause widespread **maternal endothelial cell damage**. The resulting **capillary leakage** is responsible for third spacing of fluid (eg, facial edema, weight gain) and leakage of protein into the urine (ie, proteinuria). In addition, disrupted endothelial control of vascular tone causes hypertension and **widespread vasospasm**, resulting in end-organ hypoperfusion. In the kidneys, vasospasm causes decreased renal blood flow and glomerular filtration rate, leading to **minimal, concentrated urine** (ie, high specific gravity) and increased serum creatinine levels.

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(Choice C) Inflammation of the renal interstitium (eg, acute interstitial nephritis) can cause oliguria but is typically drug related (eg, nonsteroidal anti-inflammatory drugs, beta-lactam antibiotics) and associated with rash and fever, which are not seen in this patient.

(Choice D) A stone within the renal calyx (eg, staghorn calculus) may decrease the urine output of the affected kidney due to obstruction; however, overall urine output is typically preserved due to normal functioning of the contralateral kidney. In addition, renal stones typically cause hematuria.

Educational objective:
Preeclampsia is new-onset hypertension at ≥ 20 weeks gestation plus proteinuria and/or signs of end-organ damage. Preeclampsia is caused by widespread maternal endothelial cell damage due to release of antiangiogenic factors from an ischemic placenta. The result is widespread capillary leakage (proteinuria, edema) and vasospasm (hypertension, end-organ hypoperfusion [eg, renal failure]).

A 35-year-old woman, gravida 1 para 0, comes to the office for an initial prenatal visit. The patient has had no vaginal bleeding or abdominal pain and has not yet felt fetal movement. Menarche occurred at age 13. She sometimes has heavy menstrual bleeding with passage of clots, and other times she skips her menses. The patient's estimated gestational age based on her last menstrual period is 16 weeks. She has type 1 diabetes mellitus that is controlled with insulin. She smokes a pack of cigarettes daily. As part of her prenatal laboratory screening, a second-trimester maternal serum quadruple screen is performed and reveals an elevated alpha-fetoprotein level. Which of the following is the most likely etiology of this patient's abnormal screening result?

- ☐ A. Dating error
- ☐ B. Down syndrome
- ☐ C. Edwards syndrome
- ☐ D. Fetal growth restriction
- ☐ E. Fetal heart defect
- ☐ F. Hydatidiform mole

Submit

A 35-year-old woman, gravida 1 para 0, comes to the office for an initial prenatal visit. The patient has had no vaginal bleeding or abdominal pain and has not yet felt fetal movement. Menarche occurred at age 13. She sometimes has heavy menstrual bleeding with passage of clots, and other times she skips her menses. The patient's estimated gestational age based on her last menstrual period is 16 weeks. She has type 1 diabetes mellitus that is controlled with insulin. She smokes a pack of cigarettes daily. As part of her prenatal laboratory screening, a second-trimester maternal serum quadruple screen is performed and reveals an elevated alpha-fetoprotein level. Which of the following is the most likely etiology of this patient's abnormal screening result?

- ✓

☐

A. Dating error (39%)
- ✗

☒

B. Down syndrome (28%)
- ☐

C. Edwards syndrome (7%)
- ☐

D. Fetal growth restriction (10%)
- ☐

E. Fetal heart defect (4%)
- ☐

F. Hydatidiform mole (9%)

Incorrect

Correct answer
A

39%

Answered correctly

04 secs

Time Spent

2023

Version

Explanation

Maternal serum α -fetoprotein screening	
↑ MSAFP	↓ MSAFP

Maternal serum α-fetoprotein screening	
↑ MSAFP	↓ MSAFP
<ul style="list-style-type: none">• Open neural tube defects (eg, anencephaly, open spina bifida)• Ventral wall defects (eg, omphalocele, gastroschisis)• Multiple gestation	<ul style="list-style-type: none">• Aneuploidies (eg, trisomy 18 & 21)
MSAFP = maternal serum α-fetoprotein.	

The **maternal serum quadruple screen** is used to identify pregnancies at risk for **congenital defects** or **fetal aneuploidy** (eg, Down syndrome). It measures the concentration of **alpha-fetoprotein (AFP)**, estriol, β-hCG, and inhibin A in the maternal blood.

AFP is a glycoprotein produced in the fetal liver and gastrointestinal tract. Levels are **dependent on gestational age** and maternal conditions such as diabetes mellitus (typically associated with decreased MSAFP and estriol levels). Therefore, accurate pregnancy dating and complete medical history are required to correctly interpret an AFP level.

The most common cause of an **abnormal AFP level** is inaccurate pregnancy dating (ie, **dating error**). In patients with irregular menses, dating by a last menstrual period can underestimate the true gestational age. Therefore, these patients require a fetal ultrasound, which can accurately determine gestational age and evaluate for other common causes of elevated MSAFP levels, which include multiple gestation (eg, twin pregnancy), open neural tube defects, and abdominal wall defects.

(Choices B and C) Down syndrome (trisomy 21) and **Edwards syndrome** (trisomy 18) are associated with low AFP levels.

AFP is a glycoprotein produced in the fetal liver and gastrointestinal tract. Levels are **dependent on gestational age** and maternal conditions such as diabetes mellitus (typically associated with decreased MSAFP and estriol levels). Therefore, accurate pregnancy dating and complete medical history are required to correctly interpret an AFP level.

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(Choices B and C) Down syndrome (trisomy 21) and **Edwards syndrome** (trisomy 18) are associated with low AFP levels.

(Choice D) Fetal growth restriction can occur due to tobacco use, which causes placental ischemia and resultant placental insufficiency. However, maternal serum quadruple screen shows decreased estriol levels, not increased AFP levels, with fetal growth restriction.

(Choice E) Although the risk for fetal heart defects is increased with maternal diabetes mellitus, heart defects are not associated with abnormal AFP levels because AFP is not produced in fetal cardiac tissue.

(Choice F) Hydatidiform moles and other gestational trophoblastic diseases typically present with markedly elevated β -hCG levels, not elevated AFP levels. Patients may have vaginal bleeding and a significantly enlarged uterus.

Educational objective:

The maternal serum quadruple screen assesses for risk of congenital defects and fetal aneuploidy and includes measurement of alpha-fetoprotein levels, which are dependent on gestational age. An abnormal alpha-fetoprotein level is most commonly due to a dating error (eg, inaccurate gestational age).

References

AFP is a glycoprotein produced in the fetal liver and gastrointestinal tract. Levels are **dependent on gestational**

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Exhibit Display

Trisomy 18 (Edwards syndrome)

The illustration shows a baby with several physical characteristics of Trisomy 18 (Edwards syndrome). The baby is standing, wearing a white diaper. The following features are labeled with lines pointing to them:

- Prominent occiput
- Low-set ears
- Micrognathia
- Clenched hands with overlapping fingers
- Heart defects
- Kidney defects
- Limited hip abduction
- Rocker-bottom feet

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measurement of alpha-fetoprotein levels, which are dependent on gestational age. An abnormal alpha-fetoprotein level is most commonly due to a dating error (eg, inaccurate gestational age).

References

A 41-year-old woman comes to the office after a positive home urine pregnancy test. Her last menstrual period was 12 weeks ago. For the past month, the patient has had increasing nausea. She vomits several times a day and has difficulty keeping any food down. The patient has 3 children, all born at term via cesarean section. On this visit, ultrasound demonstrates echogenic intrauterine tissue without an amniotic sac and multiple bilateral ovarian cysts. Hydropic villi are evacuated from the uterus during dilation and curettage. Which of the following should be closely monitored in this patient after the procedure?

- ☐ A. Alpha-fetoprotein level
- ☐ B. β -hCG level
- ☐ C. Carcinoembryonic antigen level
- ☐ D. CA-125 level
- ☐ E. Vaginal cytology

Submit

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- A. Alpha-fetoprotein level (4%)
- ✓

B. β -hCG level (82%)
- C. Carcinoembryonic antigen level (4%)
- D. CA-125 level (6%)
- E. Vaginal cytology (0%)

Correct

82%
Answered correctly

04 secs
Time Spent

2023
Version

Explanation

Hydatidiform mole		
Type	Complete	Partial
	<ul style="list-style-type: none">Vaginal bleedingEnlarged uterus	<ul style="list-style-type: none">Vaginal bleeding

Hydatidiform mole		
Type	Complete	Partial
Clinical features	<ul style="list-style-type: none">• Vaginal bleeding• Enlarged uterus inconsistent with dates• Extremely high β-hCG levels that can cause:<ul style="list-style-type: none">◦ Hyperemesis gravidarum◦ Preeclampsia◦ Hyperthyroidism◦ Theca lutein cysts	<ul style="list-style-type: none">• Vaginal bleeding• Crampy abdominal pain• Normal uterine size• Normal to high β-hCG levels
Pathologic features	<ul style="list-style-type: none">• Marked trophoblastic proliferation & edematous chorionic villi• Bunch-of-grapes appearance• No fetal/embryonic tissue	<ul style="list-style-type: none">• Some enlarged villi with focal trophoblastic proliferation• Fetal/embryonic tissue present
Karyotype	<ul style="list-style-type: none">• 46,XX or 46,XY (paternal DNA only)	<ul style="list-style-type: none">• 69,XXX or 69,XXY (maternal & paternal DNA)
Immunohistochemistry	<ul style="list-style-type: none">• p57 negative	<ul style="list-style-type: none">• p57 positive

Clinical features	<ul style="list-style-type: none">Enlarged uterus inconsistent with datesExtremely high β-hCG levels that can cause:<ul style="list-style-type: none">Hyperemesis gravidarumPreeclampsiaHyperthyroidismTheca lutein cysts	<ul style="list-style-type: none">Vaginal bleedingCrampy abdominal painNormal uterine sizeNormal to high β-hCG levels
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Karyotype	<ul style="list-style-type: none">46,XX or 46,XY (paternal DNA only)	<ul style="list-style-type: none">69,XXX or 69,XXY (maternal & paternal DNA)
Immunohistochemistry	<ul style="list-style-type: none">p57-negative	<ul style="list-style-type: none">p57-positive
Risk of gestational trophoblastic neoplasia	<ul style="list-style-type: none">15%-20%	<ul style="list-style-type: none"><5%

Hydatidiform moles are the most common types of gestational trophoblastic disease (GTD). A **complete mole** is composed of multiple cystic, **edematous hydropic villi** and has the macroscopic appearance of a **bunch of grapes** as a result of trophoblast proliferation. Microscopy would reveal **large, edematous villi** with decreased blood vessels, proliferation of the villous trophoblastic lining, and no fetal tissue. Ultrasound confirms lack of the fetal pole and amniotic sac, along with diffuse echogenic material in the uterus (eg, **snowstorm pattern**). Immunohistochemical stain **p57 is negative** in complete moles because of the absence of a maternal genome (the corresponding gene is paternally imprinted).

Classic clinical findings of a complete mole include first-trimester **vaginal bleeding** and an enlarged uterine size out of proportion to gestational age. **Hyperemesis gravidarum** can occur from increased β -hCG secretion by the proliferating trophoblast. Excessive amounts of β -hCG also stimulate ovarian growth and can cause the formation of **theca lutein cysts**. Preeclampsia, anemia, and hyperthyroidism can also be present with complete moles.

β -hCG levels must be monitored after uterine evacuation due to a significant risk of **malignant transformation**; rising or persistently elevated levels are a red flag for neoplastic conversion (eg, invasive mole, choriocarcinoma).

(Choice A) An elevated alpha-fetoprotein (AFP) level in a pregnant woman may suggest a fetal neural tube defect. Other possible causes include underestimation of gestational age, twin gestation, and abdominal wall defects. AFP is also a marker of yolk sac tumors of the ovaries and testes.

(Choice C) Carcinoembryonic antigen is a marker followed in patients with colorectal carcinoma and is not elevated in GTD.

(Choice D) CA-125 is an ovarian tumor marker and is not used for the evaluation of GTD. This patient's ovarian cysts are likely theca lutein cysts.

(Choice E) Vaginal cytology can be used to diagnose sarcoma botryoides (ie, embryonal rhabdomyosarcoma), which can occur in young girls (usually age <5 years). It has a jelly-like cystic appearance (ie, bunch of grapes) and arises from the bladder or vaginal mucosa. This patient's pathology is from the trophoblastic tissue in the uterus.

Immunohistochemical stain **p57 is negative** in complete moles because of the absence of a maternal genome (the corresponding gene is paternally imprinted).

Classic clinical findings of a complete mole include first-trimester **vaginal bleeding** and an enlarged uterine size out of proportion to gestational age. **Hyperemesis gravidarum** can occur from increased β -hCG secretion by the proliferating trophoblast. Excessive amounts of β -hCG also stimulate ovarian growth and can cause the formation of **theca lutein cysts**. Preeclampsia, anemia, and hyperthyroidism can also be present with complete moles.

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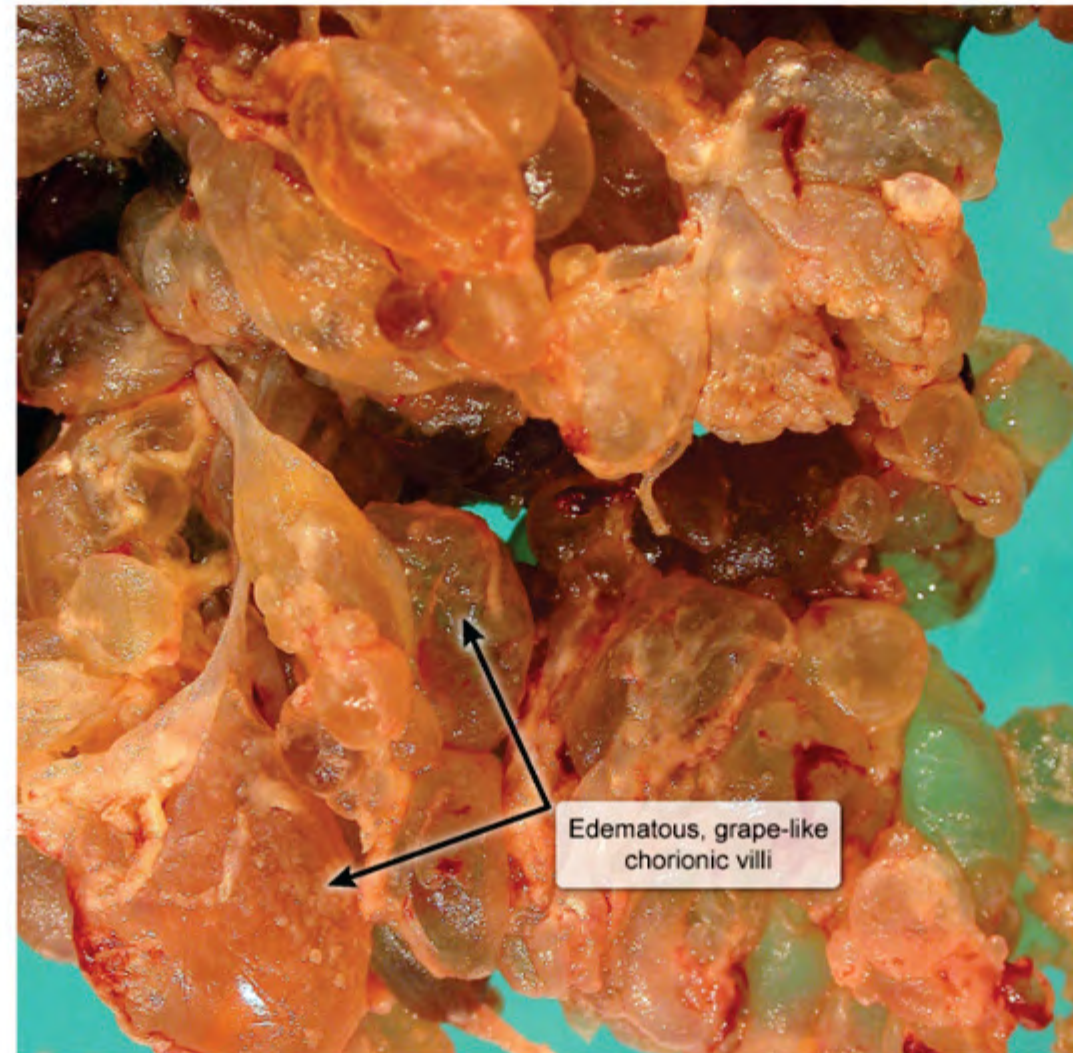
Educational objective:

A complete hydatidiform mole is composed of multiple cystic edematous hydropic villi resulting from trophoblast proliferation. Serial measurements of β -hCG should be performed following evacuation of a hydatidiform mole. Persistently elevated or rising levels may signify the development of an invasive mole or choriocarcinoma.

paternal DNA)

Exhibit Display

Complete hydatidiform mole



Edematous, grape-like chorionic villi

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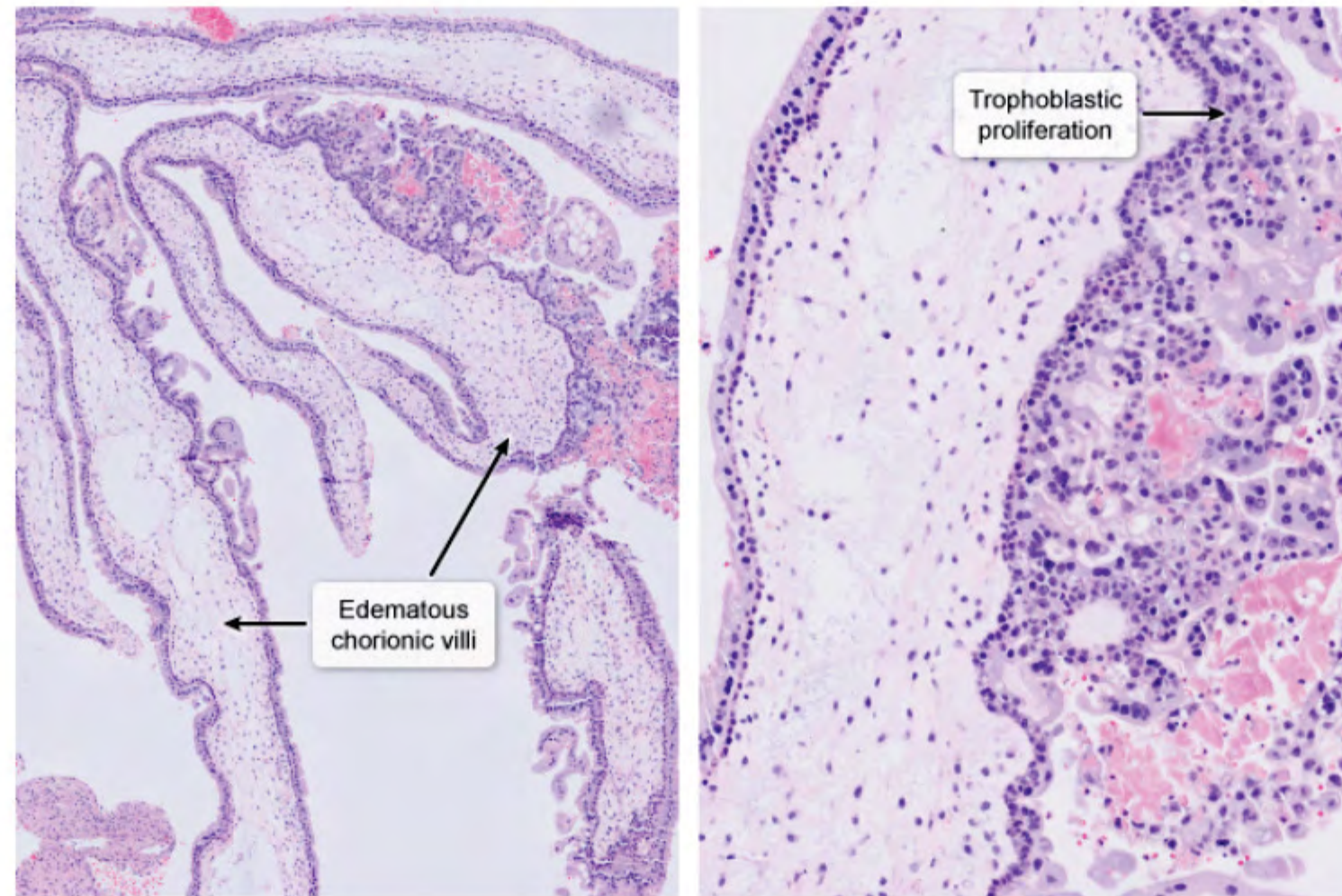
Hydatidiform mole is a gestational trophoblastic disease that can be classified as a complete or partial mole. Complete moles are characterized by the presence of numerous, edematous, grape-like chorionic villi. Incomplete moles, on the other hand, contain some normal chorionic villi along with the abnormal ones. The presence of a complete mole is often associated with elevated levels of human chorionic gonadotropin (hCG). The clinical presentation of a complete mole typically includes vaginal bleeding, uterine enlargement, and hyperemesis gravidarum. The definitive diagnosis is made through histopathological examination of the specimen.

defects. AFP is also a marker of yolk sac tumors of the ovaries and testes.

(Choice C) Carcinoembryonic antigen is a marker followed in patients with colorectal carcinoma and is not

Exhibit Display

Complete hydatidiform mole



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Exhibit Display

Complete hydatidiform mole

***Snowstorm** appearance

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defects. AFP is also a marker of yolk sac tumors of the ovaries and testes.

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A 26-year-old woman, gravida 1 para 0, at 36 weeks gestation comes to the office for a routine prenatal visit. She has had no headaches, changes in vision, or right upper quadrant pain. Fetal movement is normal. The patient has no chronic medical conditions, and her pregnancy has been uncomplicated. Blood pressure today is 150/100 mm Hg, and a repeat measurement is 154/102 mm Hg. All other vital signs are normal. Urinalysis shows 2+ protein. This patient's condition is most likely due to decreased activity of which of the following?

- ☐ A. Endothelin
- ☐ B. Human chorionic gonadotropin
- ☐ C. Prolactin
- ☐ D. Thromboxane A2
- ☐ E. Vascular endothelial growth factor

Submit

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- A. Endothelin (33%)

✖

B. Human chorionic gonadotropin (8%)

C. Prolactin (2%)

D. Thromboxane A2 (13%)

✔

E. Vascular endothelial growth factor (42%)

Incorrect

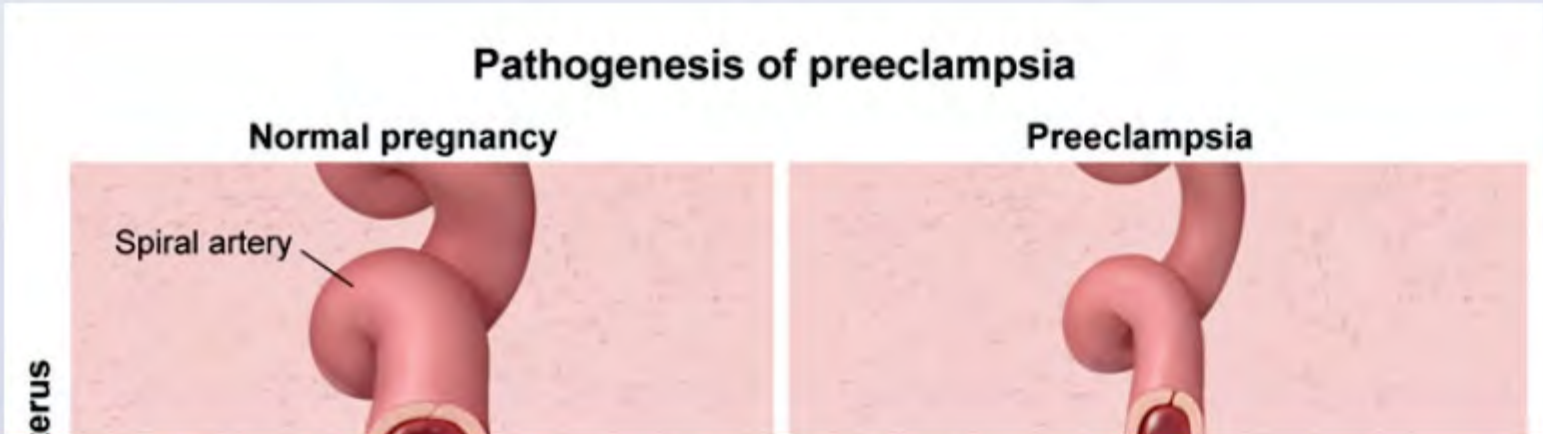
Correct answer
E

42%
Answered correctly

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Explanation



A 26-year-old woman, gravida 1, para 0, at 36 weeks gestation comes to the office for a routine prenatal visit. She

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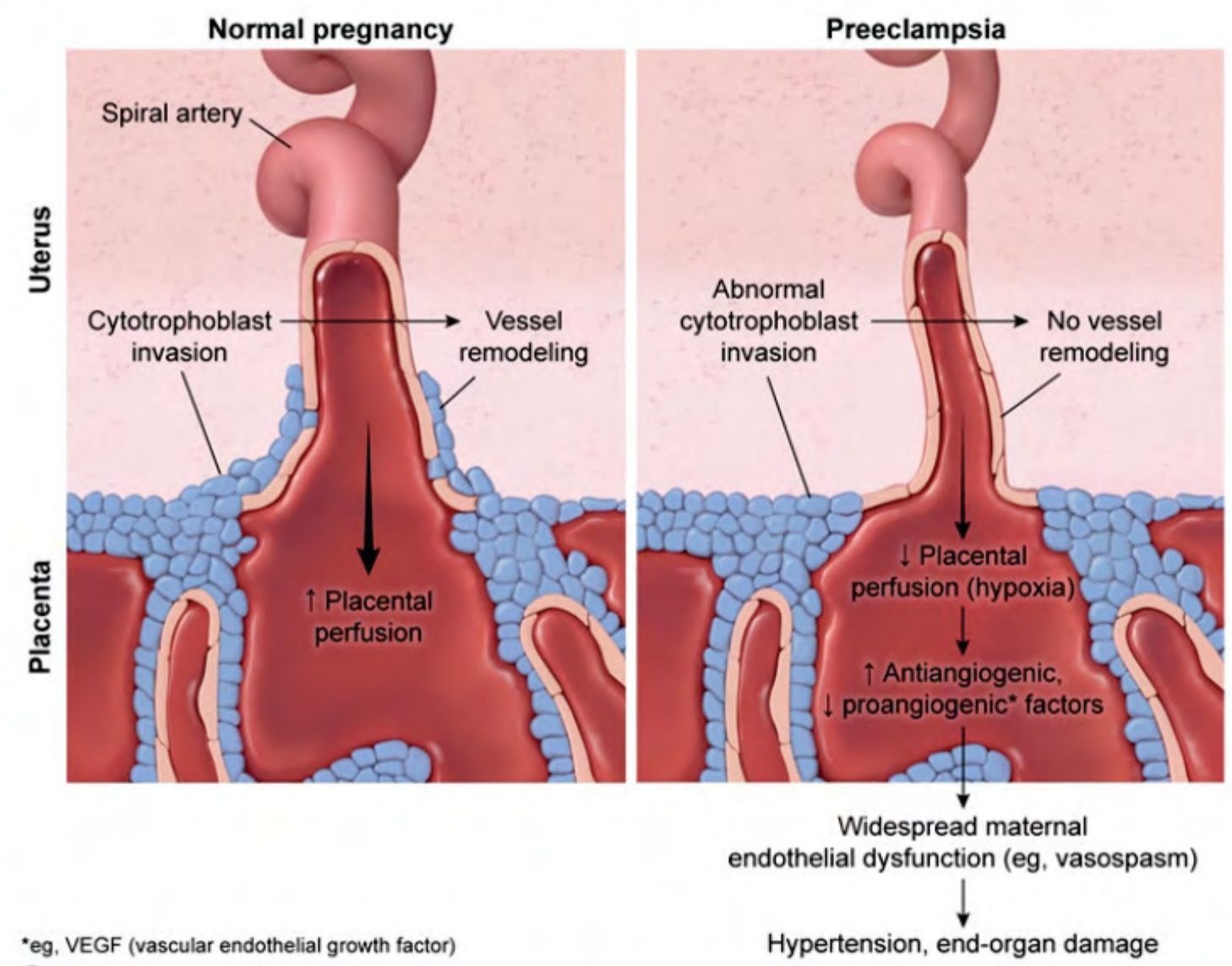
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Pathogenesis of preeclampsia



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This patient has **preeclampsia**, which is defined as new onset **hypertension** during pregnancy (systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg) with **proteinuria** and/or signs of end-organ damage (eg, headache, renal insufficiency). Although patients typically develop symptoms in the third trimester, the disease process usually begins during the first trimester with abnormal placental development.

In patients who develop preeclampsia, early cytotrophoblast invasion is abnormal; as a result, the maternal spiral arteries that supply blood to the fetoplacental unit are abnormally underdeveloped and become high resistance, low perfusion vessels. This decrease in perfusion leads to **chronic placental ischemia**, which triggers increased **release of antiangiogenic factors** throughout the maternal circulation. These antiangiogenic factors bind and **decrease proangiogenic factors** such as **vascular endothelial growth factor** (VEGF) and placental growth factor.

The overall effect is inhibited angiogenesis and widespread maternal **endothelial cell dysfunction**, resulting in dysregulated vascular tone (eg, vasospasm, hypertension), abnormally increased vascular permeability (eg, proteinuria), and decreased end-organ perfusion (eg, renal insufficiency).

(Choices A and D) Endothelin and thromboxane A2 (TXA2) are potent vasoconstrictors. Therefore, their activity is typically increased, not decreased, in preeclampsia due to increased endothelin and TXA2 production by dysfunctional endothelial cells.

(Choices B and C) Decreased human chorionic gonadotropin and prolactin levels are associated with first-trimester pregnancy loss (ie, spontaneous abortion); however, activity levels of neither hormone are related to preeclampsia.

Educational objective:

Preeclampsia is new-onset hypertension (systolic ≥ 140 mm Hg or diastolic ≥ 90 mm Hg) with proteinuria and/or signs of end-organ damage (eg, renal insufficiency). Preeclampsia is associated with widespread maternal endothelial dysfunction, which occurs due to increased antiangiogenic factor release and decreased proangiogenic factor activity (eg, vascular endothelial growth factor).

A 23-year-old woman, gravida 1, para 0, comes to the office for a routine prenatal care visit. She is at 35 weeks gestation and feels well. She reports regular fetal movement and no contractions, loss of fluid, or vaginal bleeding. Pregnancy to date has been uncomplicated and the patient has no underlying medical conditions. She signed up for a clinical trial investigating prolactin. At 34 weeks gestation, she had a serum prolactin level several times higher than what was recorded prior to pregnancy. Which of the following hormones prevents lactation in this patient?

- ☐ A. β -hCG
- ☐ B. Insulin
- ☐ C. LH
- ☐ D. Progesterone
- ☐ E. Thyroxine

Submit

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- A. β -hCG (11%)

✖

B. Insulin (0%)

C. LH (5%)

✔

D. Progesterone (80%)

E. Thyroxine (3%)
- Incorrect

Correct answer
D

80%
Answered correctly

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- Explanation
- Progesterone** and estrogen promote normal development and maternal breast growth during the prenatal period. In the first trimester of pregnancy, progesterone is secreted by the **corpus luteum** and is crucial to successful implantation. In the second and third trimesters, progesterone is produced by the **placenta** and suppresses **FSH** and **LH** through action on the anterior pituitary (**Choice C**).

Prolactin is a peptide hormone that promotes lactogenesis (ie, maturation of breast alveolar cells) and **milk**
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Progesterone and estrogen promote normal development and maternal breast growth during the prenatal period. In the first trimester of pregnancy, progesterone is secreted by the **corpus luteum** and is crucial to successful implantation. In the second and third trimesters, progesterone is produced by the **placenta** and suppresses **FSH** and **LH** through action on the anterior pituitary **(Choice C)**.

Prolactin is a peptide hormone that promotes lactogenesis (ie, maturation of breast alveolar cells) and **milk production**. It is released by the anterior pituitary gland due to stimulation by thyrotropin-releasing hormone (TRH) and increases steadily in circulation as pregnancy progresses. Although lactogenesis may begin during the second trimester, lactation is **suppressed** due to the high **progesterone** levels inhibiting the binding of prolactin to receptors on breast alveolar cells. After delivery, the precipitous drop in progesterone allows prolactin to stimulate lactation.

(Choice A) β -hCG is secreted by the embryonic **syncytiotrophoblast** during the first trimester, rising to its highest levels at the ninth week of gestation. β -hCG serves to maintain the corpus luteum until the placenta can assume responsibility for estrogen and progesterone synthesis. By mid-pregnancy, the corpus luteum degenerates and serum β -hCG decreases to a very low level. Therefore, β -hCG is not likely to have a significant effect on lactation during late pregnancy.

(Choices B and E) Thyroxine and insulin contribute to normal gestational mammary gland hypertrophy. Although high dose thyroxine can decrease the production of TRH, this patient has an elevated prolactin level which indicates that TRH production is not suppressed.

Educational objective:

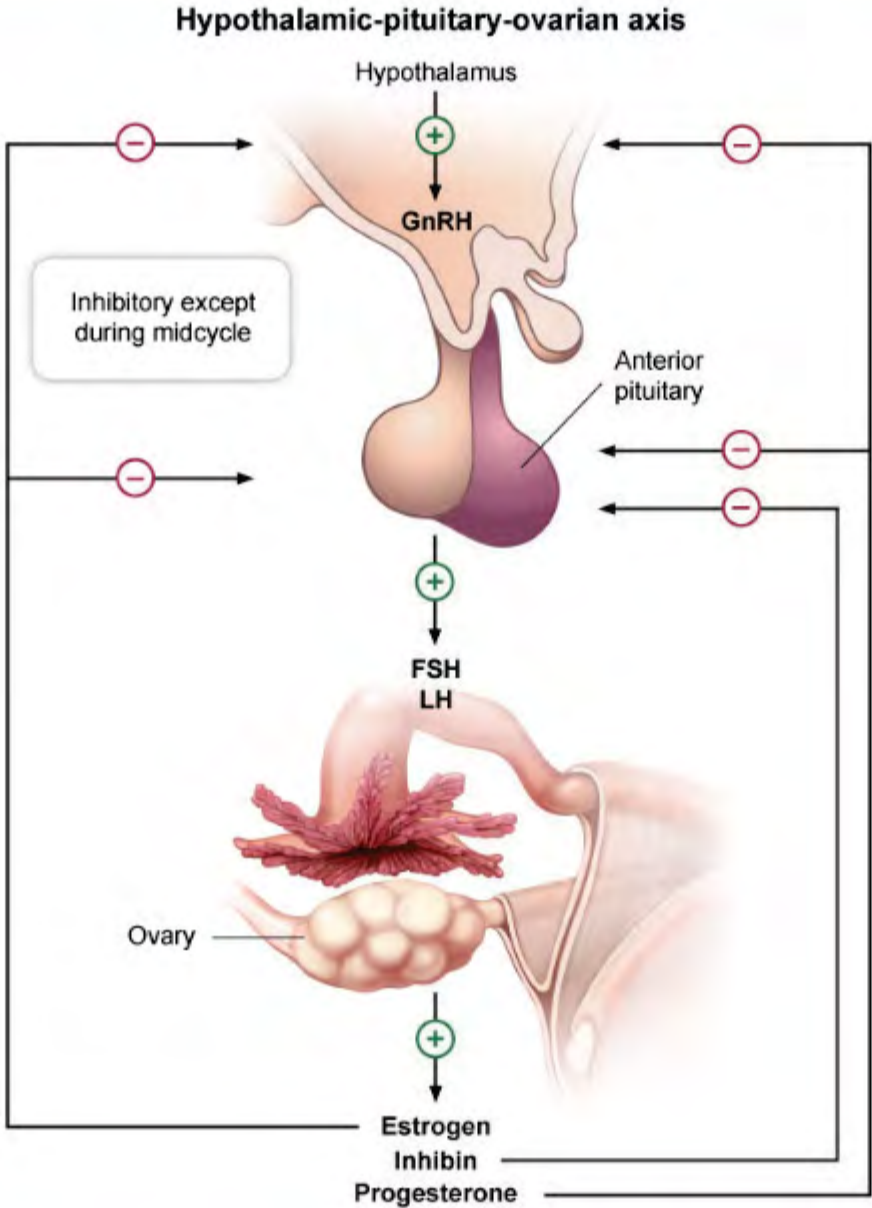
In pregnancy, progesterone is secreted by the corpus luteum and later by the placenta. Although prolactin secretion increases as pregnancy progresses, high progesterone levels inhibit lactation by preventing binding of prolactin to receptors on alveolar cells in the breast.

References

Prolactin secretion in the postpartum: mechanisms and clinical implications for milk production

Progesterone and estrogen promote normal development and maternal breast growth during the prenatal period

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References

Progesterone and estrogen promote normal development and maternal breast growth during the prenatal period

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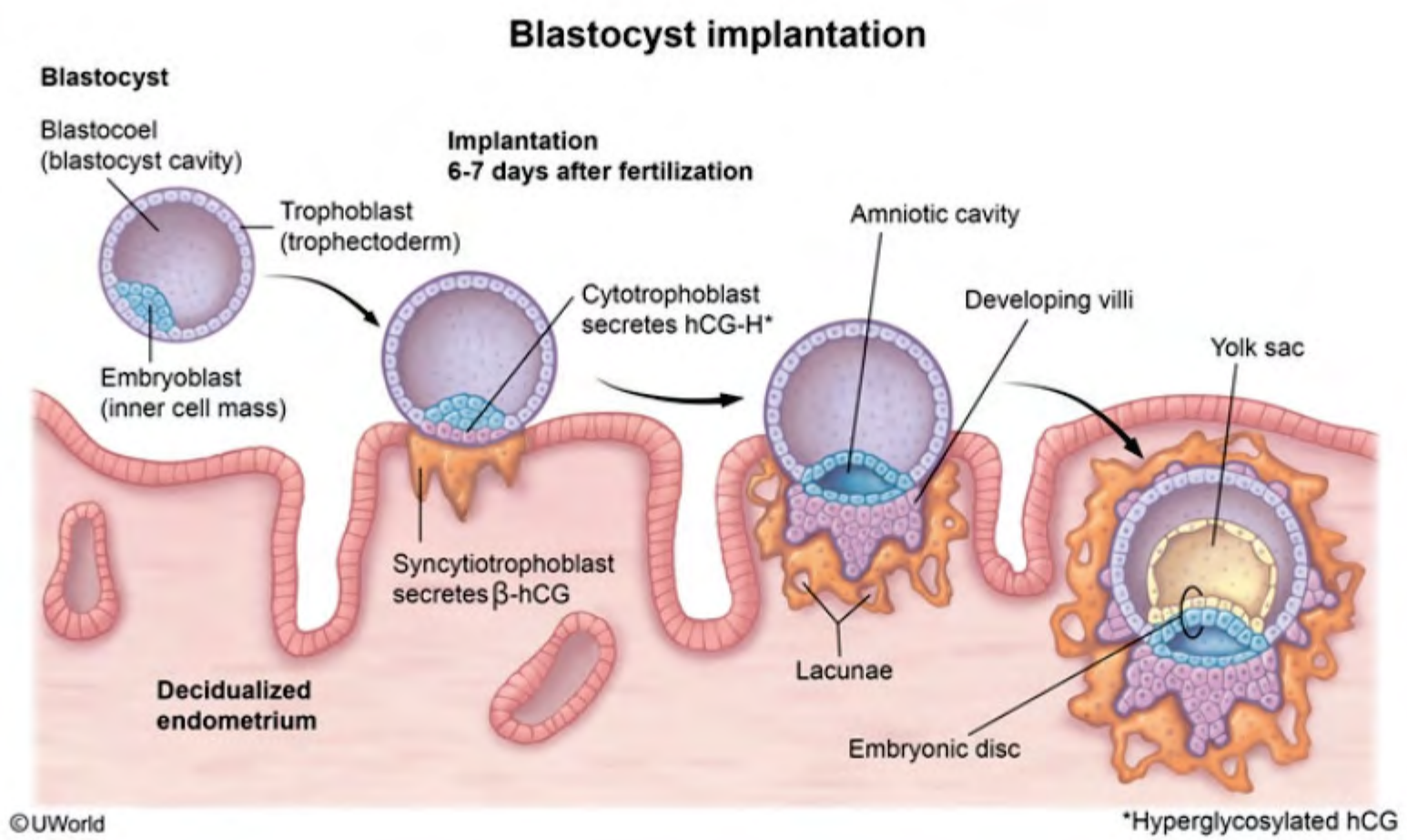
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References

Protein synthesis in the embryo is a complex process involving many different factors

A 22-year-old woman at 14 weeks gestation comes to the physician for a prenatal visit. She reports feeling well with the exception of some mild fatigue. Her pregnancy has been uncomplicated to date. On physical examination, the patient's abdomen appears larger than would be expected at 14 weeks. An obstetrical ultrasound reveals twins, a male and a female. Which of the following describes the most likely type of twin placentation in this patient?

- ☐ A. Dichorionic/diamniotic
- ☐ B. Dichorionic/monoamniotic
- ☐ C. Monochorionic/diamniotic
- ☐ D. Monochorionic/monoamniotic
- ☐ E. Monochorionic/monoamniotic conjoined

Submit

A 22-year-old woman at 14 weeks gestation comes to the physician for a prenatal visit. She reports feeling well with the exception of some mild fatigue. Her pregnancy has been uncomplicated to date. On physical examination, the patient's abdomen appears larger than would be expected at 14 weeks. An obstetrical ultrasound reveals twins, a male and a female. Which of the following describes the most likely type of twin placentation in this patient?

- ✓

☐

A. Dichorionic/diamniotic (76%)
- ✗

☒

B. Dichorionic/monoamniotic (8%)
- ☐

C. Monochorionic/diamniotic (11%)
- ☐

D. Monochorionic/monoamniotic (2%)
- ☐

E. Monochorionic/monoamniotic conjoined (1%)

Incorrect

Correct answer
A

76%
Answered correctly

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Explanation



A 22-year-old woman at 14 weeks gestation comes to the physician for a prenatal visit. She reports feeling well.

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Dizygotic twinning

The diagram illustrates the process of dizygotic twinning in two parallel vertical columns. The stages are labeled on the left: Fertilization, 2-cell stage, Morula, Blastocyst, and Embryonic disc formation. Each stage shows a corresponding biological illustration. Arrows indicate the progression from one stage to the next. At the end of the columns, two arrows branch out to show two possible outcomes: 'Separate placenta' (labeled 'Dichorionic diamniotic') and 'Fused placenta'.

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This patient is pregnant with twins of **different sexes** which can only occur in **dizygotic twins** due to the fertilization of 2 oocytes by 2 different sperm. Dizygotic twins are almost always **dichorionic/diamniotic** (eg, 2 chorions and 2 amnions) but may falsely give the appearance of being monochorionic/monoamniotic if the chorions and amnions fuse due to proximity of implantation sites.

In contrast, **monozygotic twins** develop from division of a single zygote after fertilization of a single oocyte. They are of the **same sex**, are genetically identical, and are similar in appearance. The type of placentation in monozygotic twins depends on when zygote division occurs during embryonic development.

- Early division (days 0-4) can result in monozygotic twins with 2 chorions and 2 amnions (eg, dichorionic/diamniotic), which may or may not be fused. If the sexes are the same, it may be difficult to distinguish whether the twins are monozygotic or dizygotic until later in the pregnancy.
- Division between days 4-8 is the most common outcome in monozygotic twins and results in 1 chorion (eg, shared placenta) but 2 amnions (eg, monochorionic/diamniotic twins) **(Choice C)**.
- Late division (8-12 days) results in 1 chorion and 1 amnion. A monochorionic/monoamniotic pregnancy is associated with a high fetal fatality rate, due primarily to the increased risk of umbilical cord entanglement **(Choice D)**.
- Division occurring after 13 days can result in monochorionic/monoamniotic conjoined twins **(Choice E)**.

(Choice B) Dichorionic/monoamniotic twins do not exist.

Educational objective:

Dizygotic twins occur due to fertilization of 2 oocytes by 2 different sperm, can be different sexes, and almost always have 2 chorions and 2 amnions (eg, dichorionic/diamniotic). In contrast, monozygotic twins arise from the fertilization of a single oocyte, are the same sex, and can be dichorionic/diamniotic (days 0-4), monochorionic/diamniotic (days 4-8), monochorionic/monoamniotic (days 8-12), or monochorionic/monoamniotic conjoined twins (> 13 days).

This patient is pregnant with twins of different sexes which can only occur in dizygotic twins due to the

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conjoined twins (> 13 days).

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Monozygotic twinning

The diagram illustrates the four types of monozygotic twinning based on the timing of the split and the resulting placental and amniotic configurations. The stages are categorized by time intervals: 0-4 days, 4-8 days, 8-12 days, and >13 days.

- 0-4 days:** Shows a single fertilized egg splitting into two embryos.
- 4-8 days:** Shows the two embryos developing separately, each with its own amnion and chorion.
- 8-12 days:** Shows the two embryos developing together, sharing a common amnion but having separate chorions.
- >13 days:** Shows the two embryos developing together, sharing both a common amnion and a common chorion.

The resulting configurations are labeled as follows:

- Separate placenta:** Dichorionic diamniotic
- Fused placenta:** Monochorionic diamniotic
- Shared amnion:** Monochorionic monoamniotic
- Shared chorion:** Monochorionic monoamniotic conjoined twins

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A 28-year-old woman, gravida 2 para 2, comes to the office with worsening shortness of breath over the past week. She had a recent episode of hemoptysis. The patient has also had ongoing vaginal bleeding after an uncomplicated vaginal delivery of her son 9 weeks ago. She has no bleeding elsewhere, and she has not resumed sexual intercourse. On physical examination, the patient's uterus is enlarged and the adnexa are normal. Laboratory studies show markedly increased β -hCG levels. Chest radiograph shows multiple bilateral lung nodules. Which of the following would most likely be found on endometrial curettage in this patient?

- ☐ A. Bundles of smooth muscle cells with fibrosis
- ☐ B. Diffusely hydropic chorionic villi
- ☐ C. Fetal tissue with triploid karyotype
- ☐ D. Glands lined by atypical columnar epithelial cells
- ☐ E. Proliferation of cytotrophoblasts and syncytiotrophoblasts

Submit

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- A. Bundles of smooth muscle cells with fibrosis (2%)

B. Diffusely hydropic chorionic villi (20%)

C. Fetal tissue with triploid karyotype (4%)

D. Glands lined by atypical columnar epithelial cells (6%)

E. Proliferation of cytotrophoblasts and syncytiotrophoblasts (65%)

Incorrect

Correct answer
E

65%
Answered correctly

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Explanation

Histology of gestational trophoblastic disease				
Diagnosis	Classification	Trophoblasts	Villi	Fetal/embryonic tissue
			Focally	

Histology of gestational trophoblastic disease				
Diagnosis	Classification	Trophoblasts	Villi	Fetal/embryonic tissue
Partial mole	Benign	Focally hyperplastic	Focally enlarged, hydropic	Present, triploid
Complete mole	Benign	Diffusely hyperplastic	Diffusely enlarged, hydropic	Absent
Invasive mole	Malignant	Diffusely hyperplastic with myometrial invasion	Diffusely enlarged, hydropic	Absent
Gestational choriocarcinoma	Malignant	Diffusely anaplastic/necrotic with vascular invasion	Absent	Present or absent

Gestational choriocarcinoma is a **malignant** tumor that arises from the **trophoblast**. It is most commonly preceded by a normal pregnancy but can occur following any pregnancy (molar, ectopic, aborted). The tumor causes abnormal **vaginal bleeding**, uterine enlargement, and **significantly increased β -hCG levels**.

Choriocarcinoma is aggressive and rapidly invades the uterine wall with **hematogenous** spread thereafter. The **lungs** are the most common site of distal metastasis, and presenting symptoms can include shortness of breath and hemoptysis. On macroscopic examination, gestational choriocarcinoma is seen as a bulky intrauterine mass that is usually soft and yellow-white, with extensive areas of necrosis and hemorrhage. Histologically, it is composed of abnormal proliferation of mononuclear **cytotrophoblasts** (red arrows) and multinuclear

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(Choice A) Myomas (eg, uterine fibroids) are common benign tumors of the uterine myometrium characterized by bundles of smooth muscle cells. Common symptoms include menstrual irregularities and pelvic pressure/pain.

(Choice B) Cells of the evacuated tissue of a complete mole have a 46,XX (or rarely 46,XY) karyotype containing only paternal DNA. Histologically, no fetal tissue is present, and only edematous (eg, hydropic) villi are seen.

(Choice C) Partial hydatidiform moles typically have a triploid karyotype (69,XXX or 69,XXY) containing maternal and paternal DNA, with an extra chromosome set of paternal origin. Evacuated uterine contents contain fetal tissue and other parts (eg, cord, amniotic membrane), some edematous villi with focal trophoblastic proliferation, and normal-appearing villi.

(Choice D) The normal endometrium consists of simple columnar epithelial cells. Endometrial hyperplasia characterized by crowded endometrial glands lined by atypical cells (nuclear atypia) is an important risk factor for endometrial cancer. Endometrial hyperplasia is associated with prolonged exposure to high estrogen levels (eg, obesity, nulliparity, tamoxifen use) and can present as abnormal vaginal bleeding in a postmenopausal woman.

Educational objective:

Choriocarcinoma is a malignant form of gestational trophoblastic disease composed of anaplastic cytotrophoblasts and syncytiotrophoblasts without villi. It often presents as dyspnea/hemoptysis due to pulmonary metastasis from

causes abnormal **vaginal bleeding**, uterine enlargement, and **significantly increased β -hCG levels**.

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Educational objective:

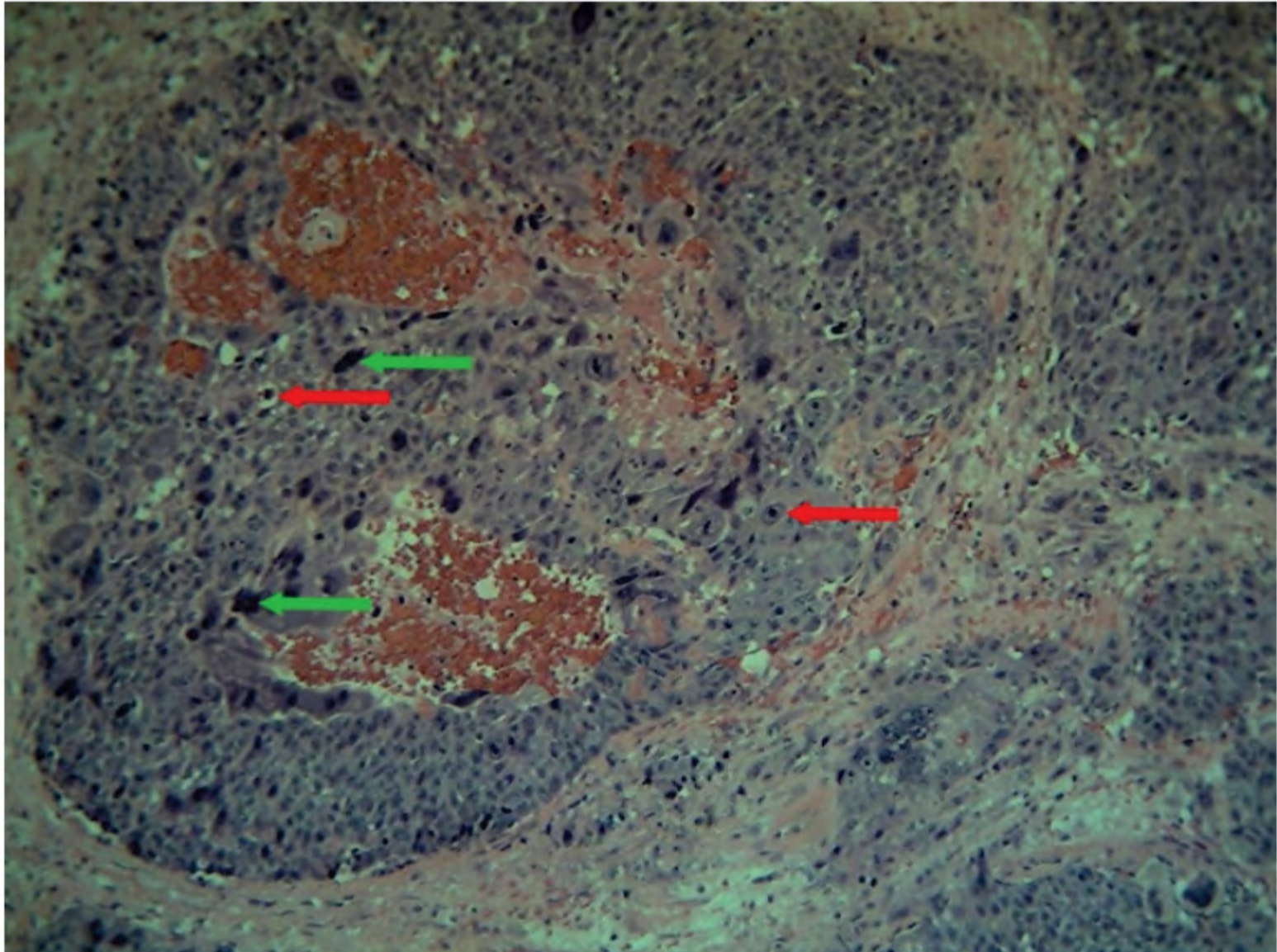
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References

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endometrial cancer. Endometrial hyperplasia is associated with prolonged exposure to high estrogen levels (eg,

A 35-year-old primigravida is found to have gallstones at 38 weeks of an uncomplicated pregnancy. Ultrasonography performed one year ago failed to demonstrate any abnormalities. Which of the following pathogenetic components most likely contributed to this patient's condition?

- ☐ A. Prolactin-induced phospholipid secretion and progesterone-induced gallbladder hypermotility
- ☐ B. Prolactin-induced sodium secretion into bile and estrogen-induced gallbladder hypomotility
- ☐ C. Progesterone-induced bile acid secretion and hCG-mediated water reabsorption from the bile
- ☐ D. Estrogen-induced bilirubin hypersecretion and cortisol-mediated cholesterol hypersecretion
- ☐ E. Estrogen-induced cholesterol hypersecretion and progesterone-induced gallbladder hypomotility

Submit

A 35-year-old primigravida is found to have gallstones at 38 weeks of an uncomplicated pregnancy. Ultrasonography performed one year ago failed to demonstrate any abnormalities. Which of the following pathogenetic components most likely contributed to this patient's condition?

- A. Prolactin-induced phospholipid secretion and progesterone-induced gallbladder hypermotility (2%)

✖

B. Prolactin-induced sodium secretion into bile and estrogen-induced gallbladder hypomotility (4%)

C. Progesterone-induced bile acid secretion and hCG-mediated water reabsorption from the bile (6%)

D. Estrogen-induced bilirubin hypersecretion and cortisol-mediated cholesterol hypersecretion (9%)

✔

E. Estrogen-induced cholesterol hypersecretion and progesterone-induced gallbladder hypomotility (76%)

Incorrect

Correct answer
E

76%
Answered correctly

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Version

Explanation

Classically, cholelithiasis (gallstone disease) is most common in those who are "fat, fertile, female, and forty." The gallstones are formed by the aggregation of bile constituents and are categorized as cholesterol stones, pigment stones, or mixed stones.

Pregnancy and the usage of oral contraceptives predispose to gallstone formation, with 5-12% of all women developing gallstones during pregnancy. Estrogenic influence increases cholesterol synthesis by upregulating hepatic HMG-CoA reductase activity, which causes the bile to become supersaturated with cholesterol. Progesterone reduces bile acid secretion and slows gallbladder emptying. When the gallbladder is hypomotile or there is more cholesterol than bile salts, the cholesterol precipitates into insoluble crystals that eventually form to

Incorrect

Correct answer
E

76%
Answered correctly

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(Choices A, B, C, and D) Cholelithiasis is not secondary to water reabsorption from bile or to the hypersecretion of phospholipids, sodium, bile acids, or bilirubin. Moreover, it is the progesterone-induced gallbladder hypomotility - not hypermotility - that increases the likelihood of developing gallstones.

Educational Objective:

Estrogen-induced cholesterol hypersecretion and progesterone-induced gallbladder hypomotility are responsible for the increased incidence of cholelithiasis in women who are pregnant or using oral contraceptives.

Pathophysiology	Pregnancy, Childbirth & Puerperium	Gallstone disease
Subject	System	Topic

A newborn is being evaluated in the nursery. The patient was born at term via spontaneous vaginal delivery to a 23-year-old woman. The mother developed dark terminal facial hair and a deepened voice during the pregnancy. The delivery was unremarkable, and the patient's vital signs are within normal limits. Examination shows ambiguous genitalia and clitoromegaly. Laboratory studies reveal elevated serum levels of testosterone and androstenedione. Karyotype testing shows a 46,XX genotype. Ultrasound of the newborn shows a normal-sized uterus. This infant's presentation is most likely due to deficiency of which of the following enzymes?

- ☐ A. 5-Alpha reductase
- ☐ B. 17-Alpha hydroxylase
- ☐ C. 21-Hydroxylase
- ☐ D. Aromatase
- ☐ E. HMG-CoA reductase

Submit

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- A. 5-Alpha reductase (5%)

✖

B. 17-Alpha hydroxylase (7%)

C. 21-Hydroxylase (48%)

✔

D. Aromatase (38%)

E. HMG-CoA reductase (0%)
- Incorrect

Correct answer
D

38%

Answered correctly

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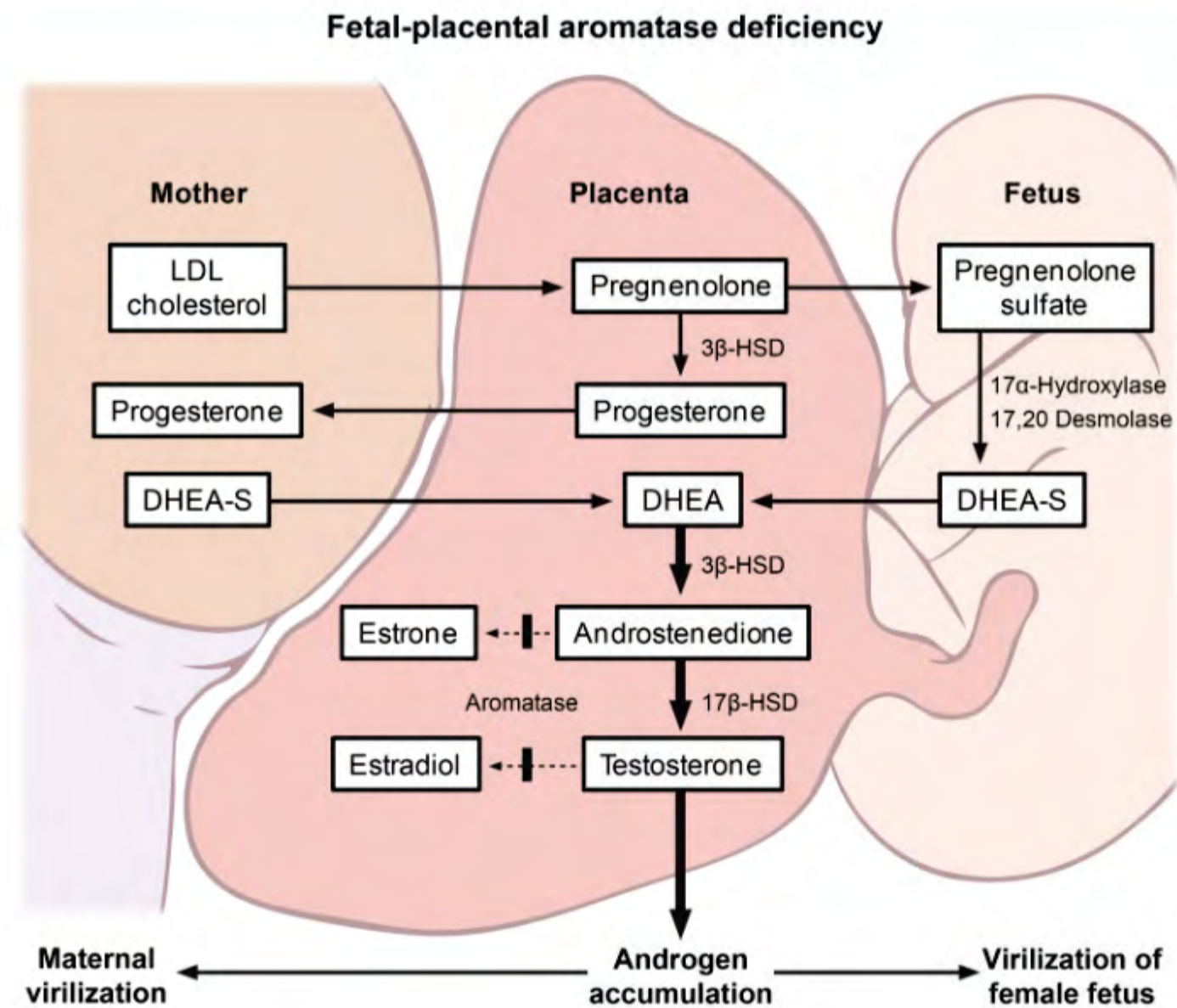
Version
- Explanation
- Fetal-placental aromatase deficiency

The diagram illustrates the concept of fetal-placental aromatase deficiency. It shows three entities: the Mother, the Placenta, and the Fetus. The Mother is represented by a large orange shape on the left. The Placenta is a red shape in the middle, connected to both the Mother and the Fetus. The Fetus is a light orange shape on the right. Below each entity is a box, likely representing a blood sample for hormone measurement. The diagram highlights that in this condition, the placenta and fetus cannot convert androgens to estrogens due to a deficiency in the enzyme aromatase, leading to elevated androgen levels in the fetus and placenta, which can cause virilization of the fetus and the mother during pregnancy.
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estrogen is synthesized by aromatase within the fetal placenta.

low estrogen) levels in the fetus and placenta. Lack of aromatase allows fetal androgens to cross the placenta and enter the maternal circulation, resulting in **maternal gestational virilization** (eg, acne, hirsutism, deep voice).

Newborn **females** with aromatase deficiency have normal internal genitalia but **ambiguous external genitalia** (eg, clitoromegaly). At puberty, estrogen synthesis is impaired due to absent ovarian aromatase, which results in primary amenorrhea, osteoporosis, and tall stature due to delayed fusion of the epiphyses. Males with aromatase deficiency have phenotypically normal genitalia but develop tall stature and osteoporosis as adults.

(Choice A) 5-Alpha reductase deficiency results in decreased synthesis of dihydrotestosterone, which is responsible for the development of external male genitalia (eg, scrotum, penis). Affected males have underdeveloped genitalia, whereas females are unaffected at birth.

(Choice B) 17-Alpha hydroxylase deficiency is a form of congenital adrenal hyperplasia. Symptoms result from decreased production of glucocorticoids and androgens (ambiguous genitalia in males) and increased production of mineralocorticoids (hypertension, hypokalemia). Females have phenotypically normal genitalia.

(Choice C) 21-Hydroxylase deficiency is the most common type of congenital adrenal hyperplasia and is associated with salt wasting and adrenal crises due to decreased aldosterone and cortisol synthesis. Increased androgen production causes ambiguous genitalia in females. Unlike this case, however, maternal virilization does not occur due to intact placental aromatase activity, which prevents androgens from entering the maternal circulation.

(Choice E) HMG-CoA reductase is the rate-limiting enzyme in the cholesterol synthesis pathway; statins inhibit this enzyme and reduce endogenous cholesterol synthesis. It is not involved in sexual differentiation.

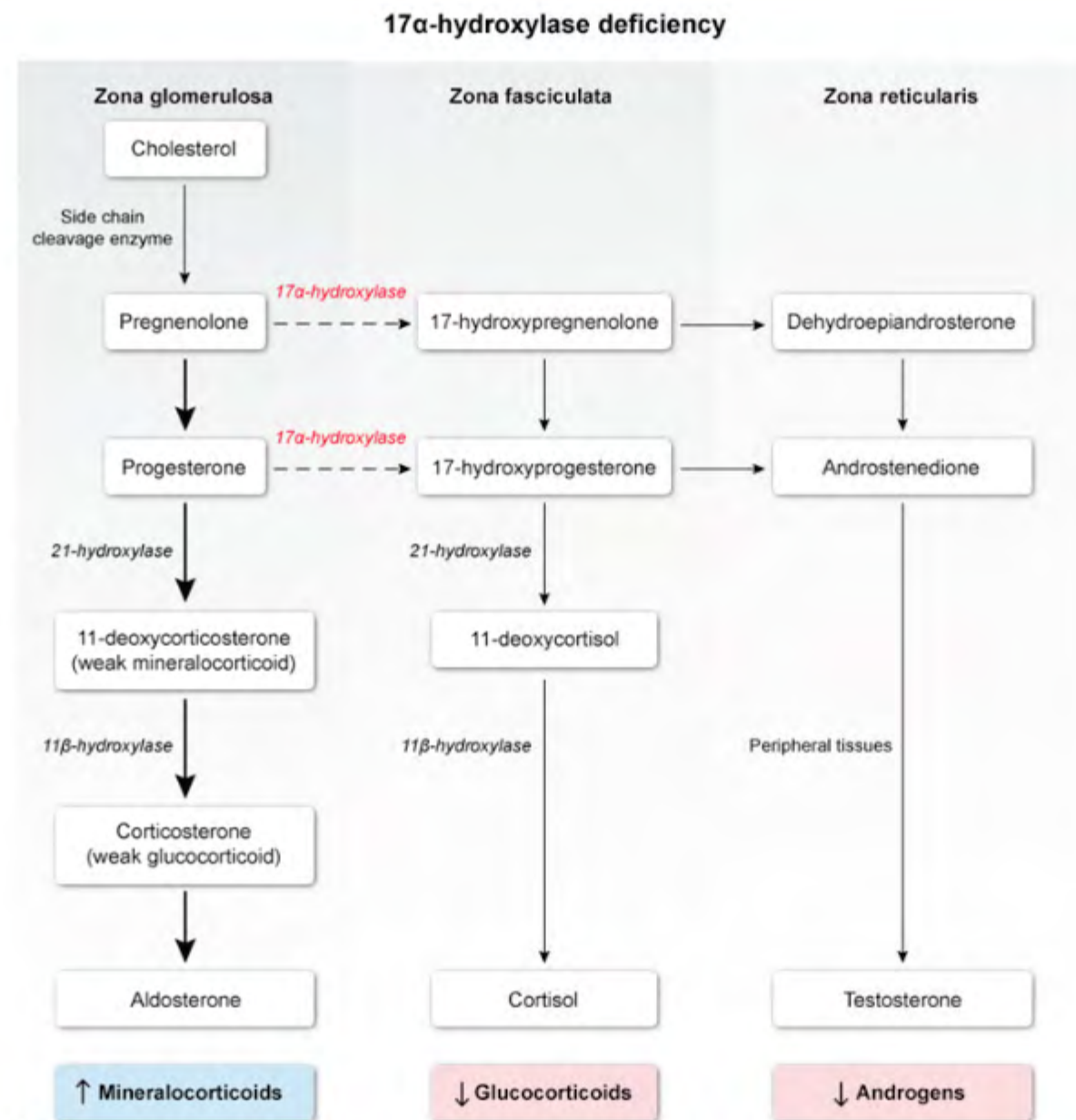
Educational objective:

Aromatase is responsible for converting androgens into estrogens. Deficiency of this enzyme causes accumulation of androgens during pregnancy, resulting in ambiguous external genitalia in newborn females and maternal gestational virilization.

References

Maternal virilization ← Androgen accumulation → Virilization of female fetus

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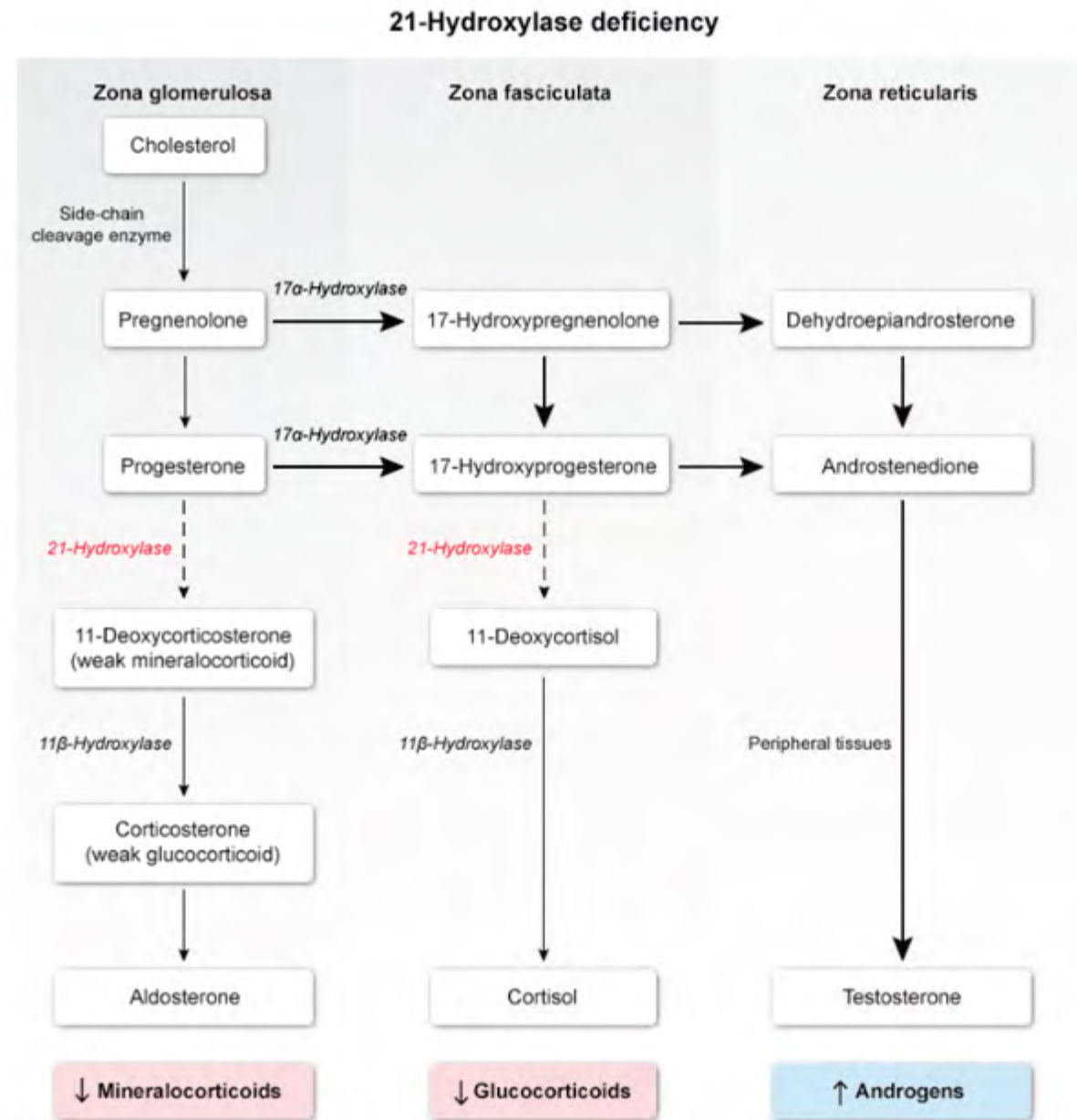


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Maternal virilization ← Androgen accumulation → Virilization of female fetus

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